THE Journal of the Kansas Medical Society

FEBRUARY
1956

VOL LVII
NO II

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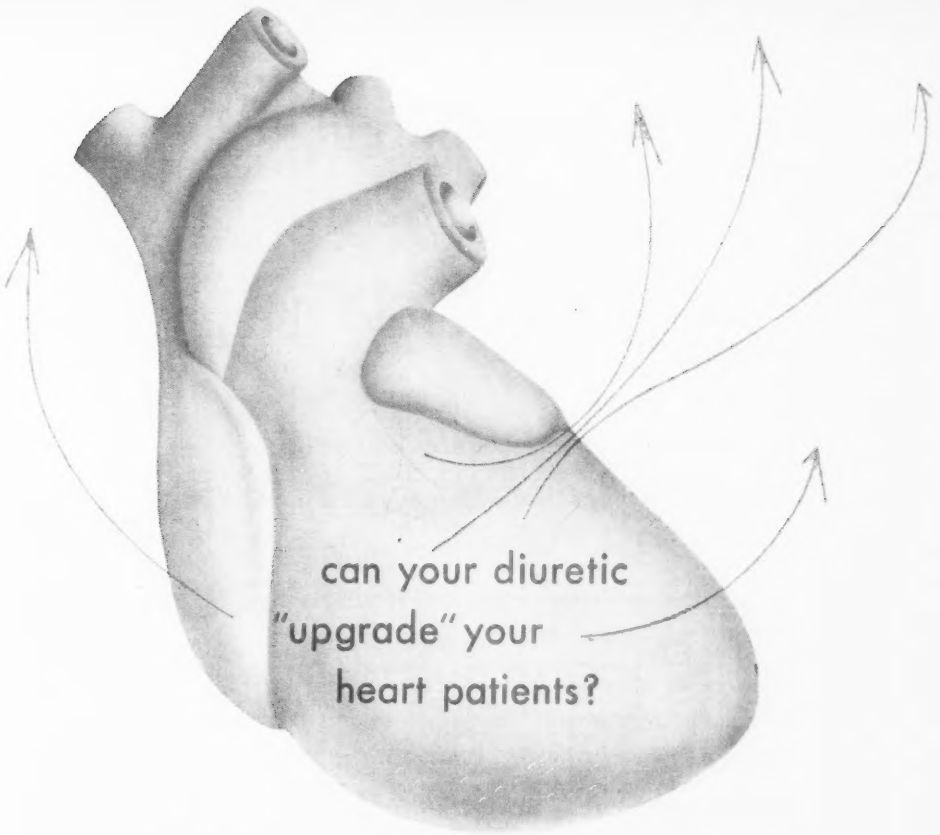

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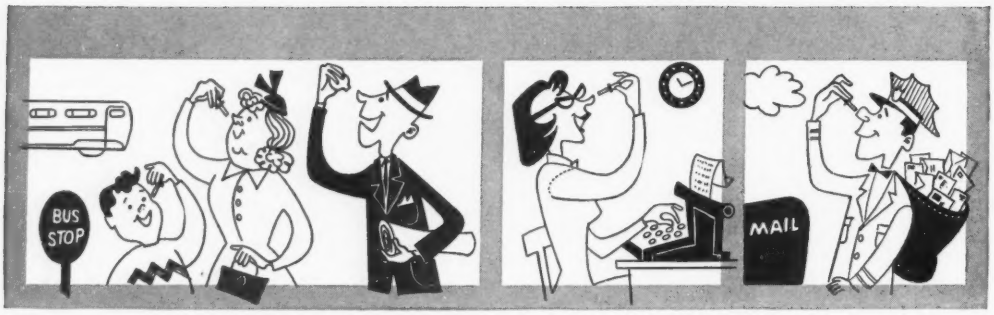

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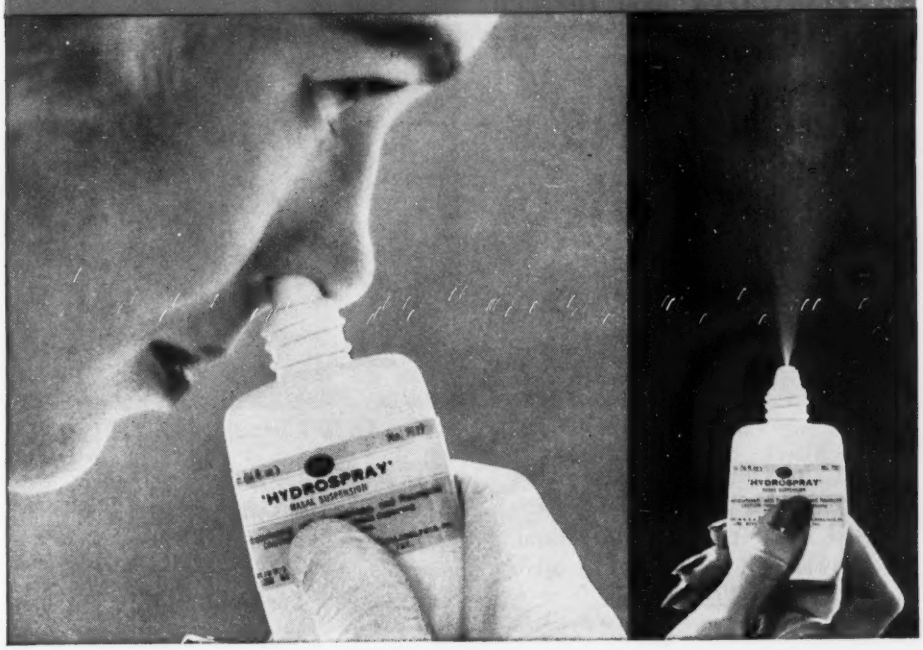

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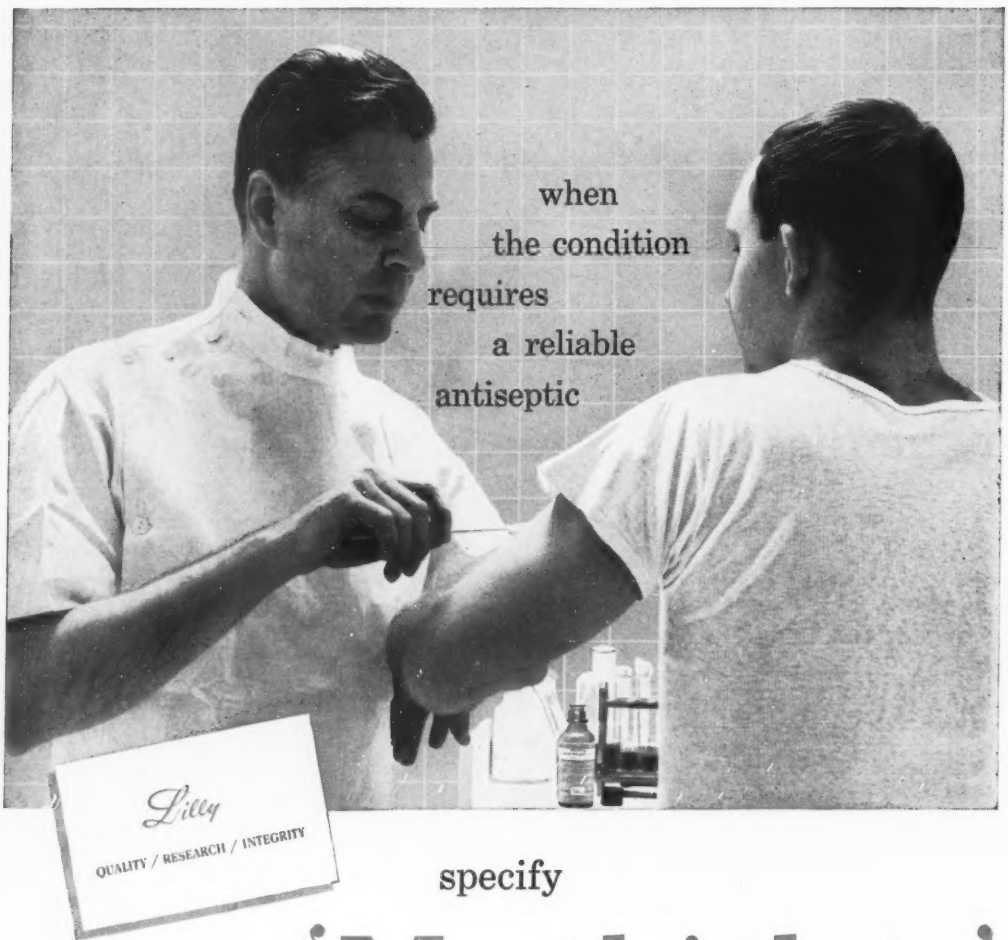

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Volume LVII **FEBRUARY, 1956** **No. 2**

Catarrhal Otitis Media

JOSEPH A. BUDETTI, M.D., ***Wichita, and***
ERNEST M. SEYDELL, M.D., ***Wichita***

Reports of ever increasing prevalence of non-suppurative fluid in the middle ear continue to be published. In all climates, catarrhal or serous otitis media is showing the same increase in prominence. The disease is described in the literature as acute, subacute, and chronic, phases which blend almost imperceptibly with one another. For the purpose of definition, however, the chronic phase may be described as the stage of desperation of the physician as the accepted remedies for the acute phase fail.

The classic explanation for fluid in the middle ear is generally accepted as follows: eustachian tube obstruction produces negative pressure in the middle ear due to absorption of air by the tissues. The negative pressure, in turn, produces vascular dilatation and transudation of sterile serum into the middle ear.

The principal etiologic factor in most catarrhal otitis medias is eustachian tube obstruction. Several principal groups of predisposing factors may be enumerated. Any lesion obstructing the ventilation of the middle ear may be the offending agent. In children, hypertrophic nasopharyngeal lymphoid tissue is probably the most common factor. Because of current success with antibiotic treatment of acute adenoid and tonsil infection, there has been a dangerous trend to procrastination of adenoid removal without regard to the long range effect on ears and hearing. The child who "won't pay attention" to his parents' call or teachers' talk, the child who keeps turning up the volume of the radio or television speaker is a real problem requiring careful study. The adult or young middle aged person who can't understand why his hearing is slipping away may have been saved this disability by prophylactic adenoidectomy in childhood.

Nasopharyngeal tumors must not be overlooked in adults. Scars or adhesions from traumatic adenoidectomies, severe posterior septal deviations, and paralysis of the tensor veli palatini muscles are among the local lesions in the nasopharynx which may cause eustachian tube obstruction.

Mandibular fractures, mal-occlusion, and overbite are dental factors which tend to cause fluid in the ear.[1] Contrary to common belief, temporomandibular joint syndrome with eustachian involvement can occur even when the rear molars are present.

A discussion of the etiology, symptoms, and diagnosis of otitis media, with suggestions for its prophylaxis and treatment.

Acute or chronic infections of the nose, sinuses, or pharynx frequently cause poor middle ear ventilation by spreading to the eustachian tube mucosa. A purulent postnasal discharge can affect the head, throat, esophagus, lungs, or stomach—why not the ears?

Perennial vasomotor rhinitis or specific nasal allergy are the most common predisposing agents in adults and may be difficult to control. The boggy tuminesence of the posterior tips of the turbinates can simulate the effect of tumor or nasopharyngeal fibromas. Endocrine factors such as hypothyroidism may be easily overlooked. An interesting point brought out by Suchs[2] is that almost all the large series of catarrhal otitis media cases are reported by physicians residing in low, humid areas. Improper nose blowing and sneezing may prolong the symptoms, if not actually precipitate them.

Low grade infections of the mucosa of the eustachian tube, frequently secondary to virus infection in the nose or throat, cause stimulation of the mucous

glands of the eustachian tube and exudation of bacterially sterile mucus into the middle ear. Explosive nose blowing may force nasopharyngeal mucus into the eustachian tube.

The apparent increase in the incidence of serous otitis media may be due to increased awareness on the part of physicians and the apparent general increase in virus infections, but it most probably is due to a relatively new phenomenon or the abortive action of antibiotics on purulent middle ear infections. Maxwell[3] recently expressed this thought quite aptly when he said, "Inadequate antibiotic therapy by means of ill chosen preparations, insufficient dosage, or premature termination of treatment may favor a smoldering chronic infection. Hearing loss in children due to low grade chronic inflammation and resulting scar in the middle ear after apparent recovery from an acute infection is an example of undesirable results which may be obtained from this type of therapy."

Schenk,[4] in describing the indiscriminate use of antibiotics in acute otitis media, said, "The ear mechanism does not necessarily return to normal merely because the fever is reduced and pain disappears." The frequency with which needle puncture-aspiration plus inflation is required in our office as part of the treatment to "open up" an ear after the infection has been overcome bears witness to the above statement.

At the 1955 session of the Kansas Medical Society, Dr. Theodore Walsh of Washington University and Barnes Hospital, guest speaker in otolaryngology, described his experience at Children's Hospital in St. Louis. As part of a recent project, 12 consecutive patients having otitis media and being discharged as cured were seen by the ENT resident for the first time on their day of discharge. All 12 drums were punctured with a needle and fluid was aspirated from the middle ear in each case. Since we know full well how some of these fluids become tenacious and adhesive, the implications of this short series can be shocking.

The symptoms of acute catarrhal otitis media are well known. There is a feeling of fullness or obstruction in the ear. Hearing loss is present and may be suddenly precipitated or relieved by yawning or blowing the nose. Head noises are frequently present and may be manifested by crackling or bubbling sounds in the affected ear. Vertigo is an occasional symptom.

Examination of the tympanic membrane may reveal extremely variable findings. The anticipated "fluid level" sign is rarely seen since most middle ears are completely filled rather than half filled with fluid. The drum is frequently retracted—even when fluid is present. Bubbles or a fluid level may sometimes be visible through the tympanic membrane. The drum may be dull grey and lusterless—or it may show the yellow-amber transparency of the accumulated fluid through its tense area. The chalk-white line of the malleus may be flattened and thinned by the fluid pressure. On occasion the drum may appear almost normal, and yet diagnostic aspiration—based on history and obstructive symptoms—will obtain fluid from the middle ear. Conduction deafness is present with lateralization of the Weber test to the deafened ear on the use of the tuning fork.

Treatment of acute catarrhal otitis media has two objectives—the removal of fluid from the middle ear and the prevention of reaccumulation of fluid by treatment of the associated predisposing condition. As a general rule, the sooner treatment is started following the onset of symptoms, the shorter will be the course of the disease. If an infection is present in the nose and throat, it should be vigorously treated. Proetz treatments using shrinking solutions are of value in shortening the course of the nasopharyngeal infection.

Vasoconstrictors may be applied to the nasopharyngeal mucosa by jetomizers, sprays, or drops. Ephedrine, 1 per cent, or neosynephrine, ¼ per cent, are the basic standards. Stronger solutions may fit appropriate cases. Ephedrine or propradrine are effective by mouth. Dilute cocaine may be applied by cotton tipped applicators directly to the tube orifices. Even vaporizers can be effective in some cases. Some writers advocate use of infra-red or short wave diathermy.

When no purulent discharge is present, eustachian tube inflations are part of the classical treatment. The Valsalva maneuver and politzeration are advocated by some. Direct catheterization of the eustachian tube has proved effective. Some writers consider it to be excessively traumatic to the eustachian cushion mucosa, and others consider it "old fashioned." In our hands it has proved to be an invaluable tool. We never hesitate to use this method and consider it far superior to Valsalva or politzeration.

The easiest and most rapid method of removal of fluid from the middle ear is by puncture of the drum with a 20- or 22-gauge needle and aspiration by any one of numerous gadgets or syringes. Some writers do a standard paracentesis with or without spot suction at the puncture site, especially when tenacious mucus is present. Inflation before puncture or aspiration helps clear the drum from the promontory of the middle ear. After puncture, it helps evacuate some of the residue of fluid. Local anesthesia is usually unnecessary but is satisfactory.

Needle puncture-aspiration is the method used in our office. It gives immediate relief which many times is permanent. If the ear canal and drum need cleaning, alcohol is used. A cocaine-camphor-carbolic tampon is applied to the drum for a few minutes.

The needle is inserted through the posterior inferior quadrant of the drum. Aspiration yields several tenths of a cc. of fluid which varies from a thin serous form to thick gelatinous form. The perforation of a drum heals in a matter of hours even after repeated punctures. Secondary infection is rare enough to be no problem. Some writers advise the insertion of a sterile cotton wick against the perforation to assist in subsequent drainage of fluid from the "mastoid cells," but this seems superfluous.

Aerotitis is the same disease as that just discussed, in a hyper-acute form. It is usually precipitated by rapid descent in aircraft in persons with obstructed eustachian tubes. The general treatment is the same as that for ordinary acute catarrhal otitis media, but immediate therapy is highly desirable. If treatment is instigated within two hours, relief is usually permanent. Treatment may be in the form of aspiration, Proetz treatment with strong vasoconstrictors, or inflations.

The best treatment is, of course, prevention. Flying should be avoided in persons with upper respiratory infections, sinusitis, hypertrophic nasopharyngeal lymphoid tissue, active nasal allergy, etc. All fliers and passengers should be adequately instructed in methods of shrinking nasal mucosa and inflating the middle ear if flying is unavoidable. The value of radium treatment to the nasopharynx was adequately demonstrated in World War II for those pilots showing recurrent aerotitis.

The transition from the acute stage to the chronic is almost imperceptible. The fluid becomes increasingly thick and tenacious, and the edema of the mucosa is replaced by fibrosis. Fibrous bands ultimately bind down the ossicles, the tympanic membrane becomes plastered to the promontory, and fibrous bands in the eustachian tube area produce strictures.

In the terminal stages, head noises may be the most distressing symptom. Conductive deafness is present and often irreversible. The tympanic membrane is dull, thickened, and retracted. It may be impossible to inflate the middle ear. In this stage, the prognosis is poor. The patient finally becomes adjusted to moderate deafness and head noises. Eustachian tube inflations, bougienage, and pneumomassage of the tympanic membrane may be done as long as the patient notices improvement after each treatment.

For the ill-defined subacute stage several other suggestions have been made in the literature. Hypertrophic nasopharyngeal lymphoid tissue must be removed by adenoidectomy and radium or x-ray therapy. This point cannot be stressed too strongly.

Nasal allergy should be controlled by desensitization, diet, etc. The incidence and influence of nasal allergies cannot be overemphasized. Dental abnormalities must be corrected. Occasional improvement has been noted following the use of autogenous vaccines made from the patient's nasopharynx. A writer recently advocated prolonged drainage of the middle ear by temporary implantation of a polyethylene tube through an ear drum puncture. Permanent perforations of the tympanic membrane have been advocated, but mastoid complication of this method must be assumed as a calculated risk. Simple mastoidectomies have been resorted to in a few cases. ACTH has been used by one of us (J. B.) successfully in a few vasomotor rhinitis cases that would not respond to other means.

In the treatment of chronic catarrhal otitis media, once again the keynote is prophylaxis. Acute attacks should be treated enthusiastically and predisposing factors eradicated whenever possible. The unhealthy tendency to postpone or avoid adenoidectomy in childhood because antibiotics cure acute attacks has been leading to many cases of potential conductive deafness in adult life. Fortunately the pendulum is swinging back to a sensible mid-point as most physicians realize the inherent dangers of chronic or subacute recurrent infections. Infections are cured but the congestion and hypertrophy remain to impinge on the eustachian tubes and predispose the "cured" child to premature deafness in his third and fourth decade of life. Tonsil and adenoid surgery is still the physician's best weapon for this prophylaxis.

104 West Douglas
Wichita 2, Kansas

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Every man has two educations—that which is given to him and, the other, that which he gives himself. . . . Indeed, all that is most worthy in a man he must work out and conquer for himself. It is that which constitutes our real and best nourishment.

—*Jean-Paul Richter*

Bronchography

Use of Aqueous and Oily Suspensions of Dionosil

ALFRED M. TOCKER, M.D., *Wichita*

The superiority of iodized oil (Lipiodol) as a medium for bronchography has been virtually unchallenged since its introduction in 1922.[20] However, the substance has a number of disadvantages. The more important of these are alveolar filling which often interferes with interpretation of the bronchogram, persistent visibility in the alveoli seen long after instillation and often interfering with interpretation of future chest roentgenograms, and liability to produce iodism.[22]

Because of these disadvantages of Lipiodol, other media, in particular rapidly absorbing radiopaque water-soluble media, were developed. These, however, proved to be cough-provoking bronchial irritants necessitating tedious and time-consuming anesthetic techniques, with the inherent dangers of the anesthetic agents, and often resulted in unsatisfactory bronchograms.[6, 7, 14, 16] As a result, the preference of Lipiodol as a contrast medium for bronchography continued unchallenged.

In 1952 a new type of contrast medium, Dionosil, was developed in England.[4, 23] Reports from all sections of the world have since indicated that this contrast suspension is replacing Lipiodol to a large extent in many medical centers.[1, 7, 10, 15, 24] Since its introduction into the United States in 1954, it has gained great popularity. After careful evaluation of 35 cases in which bronchography was performed with Dionosil,* I have come to use this product exclusively for bronchography, although it is conceivable that there are special cases in which Lipiodol would be the preferable agent.

Dionosil differs from other media in that it is a suspension (rather than a solution or iodized oil) of the n-propyl ester of 3:5-di-iodo-4-pyridone-N-acetic acid (propyliodone).[23] It is presented in two forms —aqueous (a 50 per cent w/v suspension in a buffered aqueous vehicle containing sodium carboxymethylcellulose as a suspending agent) and oily (a simple 60 per cent w/v suspension in peanut oil). Preference among our roentgenologists varies between the more viscid oily suspension and the slightly more irritating aqueous suspension, the latter being favored in most cases. Both forms are less viscous than Lipiodol.

In Dionosil the deficiencies of iodized oils have been overcome as follows: the ester is completely hydrolysed, absorbed into the blood stream, and excreted in the urine within a few days; and degradation does not extend to the liberation of iodides or iodine which might give rise to sensitivity reactions.[23]

Dionosil, a recently developed bronchographic medium, is a safe contrast suspension with attributes at least equivalent to those of Lipiodol, with the significant advantages of rare alveolar filling and rapid disappearance from the lung.

Advantages of Dionosil include the following:

1. *Rapid Disappearance from Lungs.*[1, 4, 7, 10, 13, 17, 18, 19, 23, 24] Dionosil is eliminated by drainage, cough, and excretion through the kidneys[23]—often within 24 hours, and completely in a few days (Figure I). Bronchography may be repeated at any time.

2. *Infrequent Alveolar Filling.*[1, 2, 4, 8, 9, 18, 23, 24] Significant alveolar filling is unlikely, even if coughing occurs. When alveolar filling does occur, absorption is not delayed. In two of our cases, alveolar filling persisted in pathological areas which were later removed surgically and diagnosed microscopically as showing chronic organizing pneumonia.

3. *Improvement in Detail.*[2, 4, 10, 19, 21] The suspension is deposited on mucosal surfaces, outlining rather than filling the bronchi, thus giving superior visualization.

4. *Minimal Irritation.*[3, 4, 8, 9, 11, 15, 23, 24] The oily suspension of Dionosil has almost no irritation; an aqueous suspension is slightly more irritant. Bronchoscopy by others[3] and by me within a short time after the use of Dionosil revealed no evidence of irritation. Microscopic examinations of lung tissue excised shortly after bronchography with this contrast media have shown no deleterious effect.[3, 9, 24] While some feel there is no need to postpone operation after bron-

* Prior to approval by the Food and Drug Administration for sale of Dionosil in the United States, the oily and aqueous Dionosil used in these cases was supplied to us for clinical study by Glaxo Laboratories, Ltd., of England through Picker X-ray Corporation, White Plains, New York.

The material in this article was presented in exhibit form at the Kansas Medical Society meeting in Hutchinson, May 3-5, 1955.

chography in these cases,[3, 4] others,[9] including this author, prefer to wait until the lung is clear of the contrast material.

5. *Ample Exposure Time for Films.*[21] Shadows remain well defined for at least 30 minutes; thus the need for spot-films is eliminated. However, during this time the patient must not be permitted to cough vigorously.

6. *No Risk of Iodism.*[1, 4, 9, 23] There is no breakdown to iodides or iodine in the body. The compound is completely hydrolyzed and eliminated entirely by the kidney. Iodine and iodide ions are absent in the urine.[23]

7. *Safety in Tuberculosis Cases.*[4, 5, 8, 12, 17, 19] Experience confirms that, provided the infection is controlled, use of Dionosil in bronchography is reliable and safe even in active tuberculosis cases with cavitation and positive sputum.

8. *Introduction by Any of the Presently Recognized Routes.*[11] The aqueous suspension may be introduced through an endotracheal catheter, by needle puncture of the cricothyroid membrane, or through the tracheal wall below the cricothyroid cartilage. The oily solution may be introduced by any of the above routes, as well as over the tongue.

9. *No Necessity for Special Radiographic Facilities.*[3, 4, 7, 8, 9, 16, 18, 24] The density of the compound permits satisfactory fluoroscopic examination and enables excellent films to be obtained with the usual exposures.

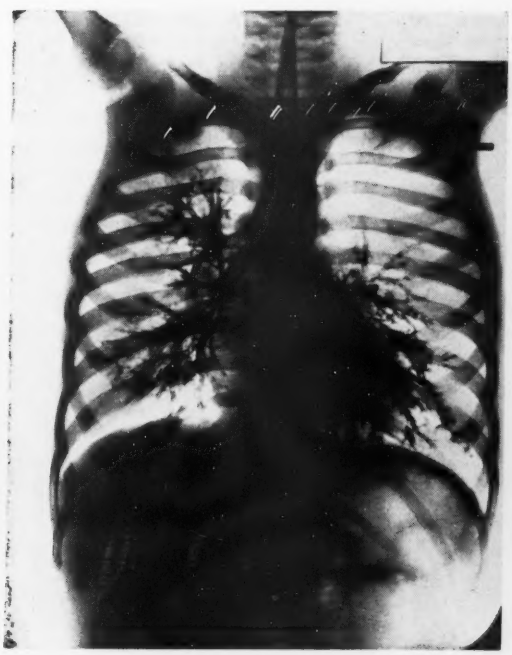
10. *Increased Scope of Bronchography.*[3, 4, 9] Limitations imposed by earlier contrast media are removed.

The disadvantages of Dionosil, relatively few, include the following:

1. *Careful Anesthesia Required.*[10, 18, 21] The suspensions (especially aqueous Dionosil) are slightly cough-provoking. In cases in which bronchoscopy and bronchography are indicated, we prefer cocaine topically as the anesthetic agent, anesthetizing both main stem bronchi with a 4 per cent cocaine spray through the bronchoscope. Immediately following bronchoscopy, the contrast material is introduced through a catheter passed through the nose into the trachea, which is less cough-provoking than a catheter passed orally.

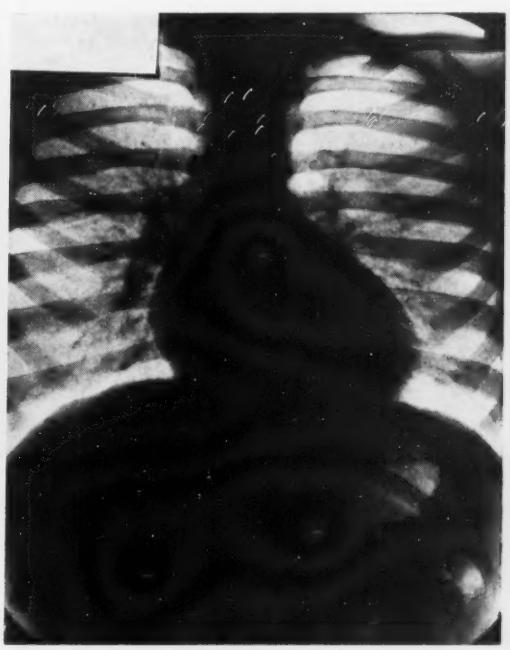
2. *Technical Difficulties in Handling Media.* The thickness of the suspension offers some technical difficulties. This is especially true of the oily suspension. The aqueous suspension, if permitted to dry, may "cake" on the tip of the syringe, interfering with injection.

3. *Pyrexial Reaction.*[1, 4, 13] This occasionally follows the use of Dionosil, but is a comparatively rare



A

(A) Bilateral bronchogram of child with aqueous Dionosil showing excellent bronchial visualization.



B

(B) PA chest roentgenogram of same child taken 24 hours after bronchography showing lungs to be substantially clear of contrast medium.

condition. In one of my cases, the patient's elevated temperature fell to normal immediately after he coughed up a small white "plug," assumed to be Dionosil.

4. *Dyspnea.*[1, 8] We believe that dyspnea has resulted in those patients in whom Dionosil was permitted to pool in the trachea or main bronchi, obstructing the respiratory passages. Our most severe reaction was in a physician who was dyspneic and complained of chest pains for several days following the bronchographic study.

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If you are to find rewarding satisfaction in your work, if your life is to be rich and purposeful and crowned with high achievement, it is important you continue to be a growing person. Education is a continuing process. It does not end with the termination of your schooling. Education continues from the beginning of life to the end of life, and balanced growth throughout one's entire life is important for every individual.

—Henry T. Maschal

Solitary Mesothelioma

A Case Involving the Pleura

ALBERT E. BAIR, M.D., *Wichita,* WILLIAM J. REALS, M. D., *Wichita, and* PAUL H. WEDIN, M.D., *Wichita*

Mesothelial tumors are an interesting though not fully understood group of tumors. Correlation of the microscopic structure and the clinical course may be difficult. A case of solitary mesothelioma of the pleura is reported, and the literature is briefly reviewed.

Mesothelial tumors have been recognized as a distinct entity since 1936. Geschickter[1] used the term for a group of tumors previously known by 30 different names, e.g. endothelioma, celiothelioma, fibroma, fibrosarcoma, leiomyosarcoma, giant cell sarcoma of the pleura, endothelial sarcoma, and so on. The embryologic origin of mesothelium has been described elsewhere.[2]

Studies made in 1942 by Stout and Murray[3] on mesotheliomas of the pleura served to fix the genesis of these tumors as mesothelial when Murray was able to explant the tumor in vitro and demonstrate that its cells imitated the behavior of normal mesothelial cells in vitro.

Three varieties of mesothelioma have been described.[4]

1. Malignant growths which are diffuse, grossly spreading, and microscopically characterized by anaplastic mesothelial cells forming tubules.

2. Solitary fibrous mesotheliomas. These may be pedunculated intrapleural growths or intrapulmonary with broad pleural attachments. Microscopically the tumor cell is elongated and spindle shaped and is associated with collagen and reticulin fibers. The arrangement is usually without definite pattern. There may be areas of increased vascularity, and when degeneration affects the tumor the collagen fibers are few. The intercellular spaces may be widened by fluid, and the cells may be rounded or stellate. In those considered malignant the histopathology reveals anaplasia. The cells are larger, stain more deeply, and mitotic division is more common. There are no collagen fibers and only extremely delicate reticulin fibers between the individual cells.

3. Benign mesothelioma of the male and female genital tracts. These are small nodules composed of tubules lined by cells that secrete a mucoid substance. There is controversy as to whether these are mesotheliomas or adenomas, adenomyomas or lymphangiomas.

Our attention was drawn to the second group by a case cared for by one of us (P.H.W.).

The patient, Mrs. J. S., a 56-year-old white female housewife, was admitted to the surgical service of Wichita-St. Joseph Hospital on October 11, 1953. Chief complaint was that of having a chest tumor. During a routine chest survey by the mobile photofluoroscopic unit of the Kansas State Board of Health, a chest tumor had been found. The family physician

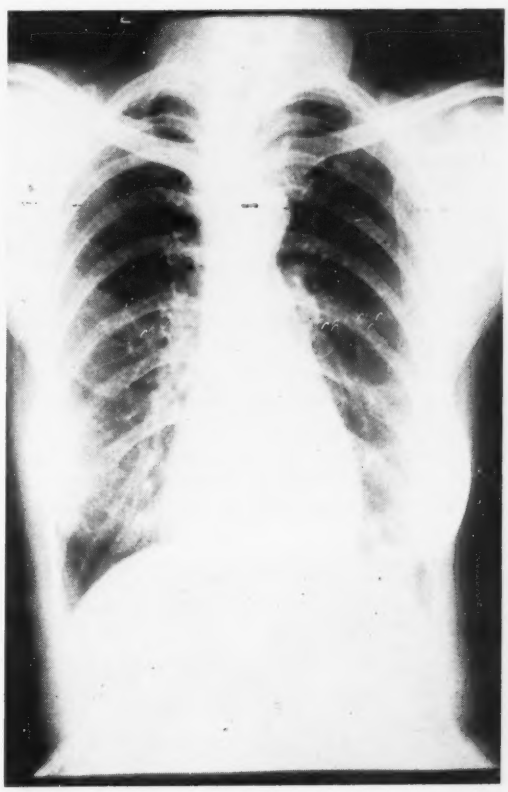

Figure 1. The shadow of the tumor is seen at the left base, occupying most of the space between the heart and the chest wall in the antero-posterior view. Lateral views showed it to be in the anterior portion of the chest.

had recommended immediate hospitalization and possible surgical exploration. Physical examination revealed no significant findings. Careful physical examination of the chest revealed no abnormalities.

The initial laboratory studies revealed a total erythrocyte count of 4,370,000 with 12 grams of hemoglobin. The differential count was 72 per cent polymorphonuclear leukocytes and 28 per cent lymphocytes. Urinalysis was negative and a VDRL test for syphilis also was negative. X-ray examination of the chest on October 11, 1953, revealed a 2.5 cm. discrete nodule in the left basal area (Figure 1). The mass was seen to lie forward on lateral views and had no association with the pericardium. The lung fields were otherwise clear, and the heart was not enlarged. The radiologist concluded that the nodule was of indeterminate nature, primary alveolar neoplasm or granuloma to be excluded.

On October 12, 1955, the patient was subjected to an exploratory thoracotomy. The left thorax was opened through the fifth interspace. When the lung was exposed a tumor was seen attached to the inferior surface of the pleura of the left lower lobe. A wedge excision was carried out. Frozen section was reported as "benign." The wound was closed in a routine manner with an indwelling catheter for drainage. On the sixth postoperative day following an uneventful recovery the patient was dismissed.

Pathological examination of the material revealed an oval mass which measured approximately 4 x 3 x 2.5 cm. The outer surface was covered by a rather thick capsule, and attached along one margin was a small appendage which consisted of pulmonary tissue. The mass appeared to be arising directly from the surface of the lung and apparently involved the pleura. On section the cut surface of the mass was light red to pink in color and whorled. Tissues on section were rubbery and cut with increased resistance (Figure 2).

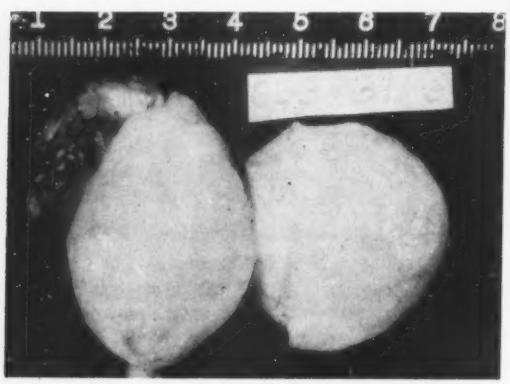

Figure 2. Gross appearance of the excised tumor, after it was divided. The cut surface was light red to pink in color, and the tumor had a rubbery consistency.

Microscopic sections of the tumor removed from the pleura showed a cellular mass composed throughout of spindle shaped cells arranged without definite pattern. In many areas the tumor was producing collagen, and in these zones the cells were scattered throughout the collagen. In other areas the collagen was relatively scanty and the nuclei were prominent. Irregular spaces which varied in size were seen, and these were lined by spindle cells. In one area there appeared to be hyalin-like changes. Evidence of epithelial cells was not found (Figure 3).

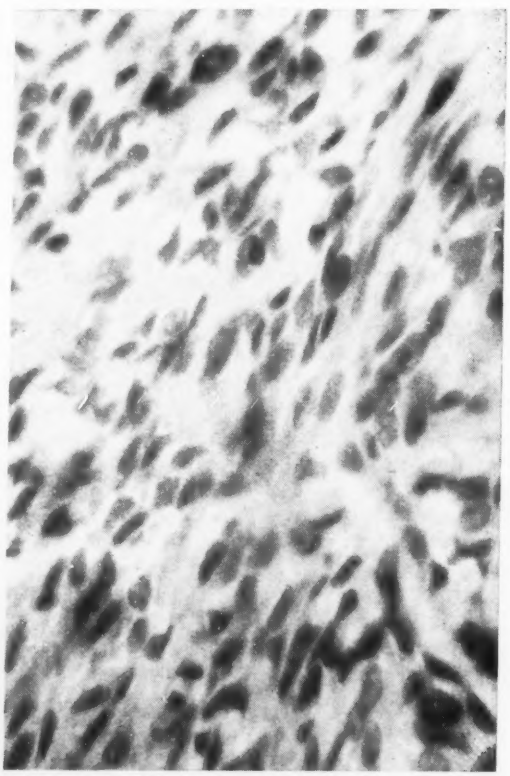

Figure 3. Photomicrograph of the tumor showing spindle-shaped cells, with no definite pattern for their arrangement. (x 740)

Diagnosis: Solitary mesothelioma of pleura.

The material was examined by Dr. Arthur Purdy Stout[5] who concurred in the diagnosis.

The patient was seen October 10, 1955, and was in good health.

Stout and Himadi[6] have reported eight cases of solitary localized mesotheliomas of the pleura, three of which were considered benign and five malignant.

Their longest period of observation was five years.

To these Benoit and Ackerman[7] have added six cases diagnosed as benign. Their longest period of observation was eight years. They have also included in their analysis three cases of Brown and Johnson,[8] all of which have had a benign course, one patient still living at 20 years. It should be noted that the latter authors considered their three cases as originating as a consequence of organization of interlobar effusions. They felt that two types of reacting cells entered into the genesis of their tumors, fibroblasts and mesothelial cells, both participating and either dominating the final picture to produce a variety of patterns.

Bogardus et al.[9] report four cases, three of which were solitary. Periods of observation have not been long enough to evaluate clinically. Two of these were microscopically malignant and one was benign.

Clagett et al.[10] have reported 24 cases of localized mesothelioma and do not feel that they have had, as yet, enough experience to classify them as benign or malignant. They reported one patient dying of recurrence five years after operation and one dying ten years after operation.

These authors add to the usual microscopic description their most significant finding, the presence of spaces or gland-like cavities lined by spindle cells.

In our review of the recent literature we have gained the impression that reported observations of patients having solitary, localized mesotheliomas have not been for a sufficient length of time to correlate with any degree of accuracy the pathological picture and the final clinical outcome. We, therefore, decline to classify our case as benign or malignant until it has stood the test of time, even though it readily fits the description both clinically and morphologically of those tumors considered benign.

CONCLUSIONS

The classification of solitary, localized mesothelioma as to its state of benignancy or malignancy is as yet a precarious undertaking. The term "five-year cure" cannot be taken as a valid criteria for the favorable outcome of any specific case.

Pathologic findings both grossly and microscopically have not been found to correlate to a great degree of accuracy with the final outcome of solitary mesothelioma of the pleura.

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All of us are here today, alive, because at many junctures in history our forefathers—undismayed by the particular fears which haunted their times—took courage from faith and transmitted faith down through succeeding generations to us. . . . Only faith is truly and invincibly strong and viable. —*Lewis L. Strauss*

Fibroma of the Spermatic Cord

MARION A. THROCKMORTON, M.D., *Halstead*

Fibromas of the spermatic cord are rare tumors, this being the 35th one reported. Since 30 per cent of spermatic cord tumors are malignant, and sarcomatous changes may develop in benign lesions, surgical removal is advised for all such tumors.

No fewer than 32 different histologic types of spermatic cord tumors have been described in the literature.[1-20] It is important for the clinician to be aware of the general agreement that about 70 per cent of all solid tumors of the spermatic cord are benign. The fact that 30 per cent prove to be malignant constitutes rational grounds for the complete removal of any cord tumor.[13, 20]

Including 1954, Gray and Biorn[20] found a total of 292 cases reported in the literature and added one of their own; 83 were malignant (28 per cent). Thirty-four simple fibromas have been reported. Morehead[9] found fibromas second only to lipomas in frequency of occurrence. They are usually small, but occasionally they attain considerable size.

CASE REPORT

A 38-year-old white male complained of a mass in the left scrotum of which he had been aware two and a half years. It was symptomless but had enlarged. He enjoyed good health, and physical examination revealed no significant disease. On the left spermatic cord was found a small discrete, firm mass which was estimated to measure 6 mm. across its ovoid surface. It felt smooth and was not attached tightly to surrounding structures. It did not transilluminate light. No lymph nodes were felt or abnormalities seen in roentgen examination.

A preoperative diagnosis of "cord tumor" was made, and the patient was advised to have the tumor removed. This was done the day following examination, using local anesthesia. A firm, glistening tumor was shelled out of the cord without bleeding. The postoperative course was uneventful.

The pathological report (Dr. C. A. Hellwig) was as follows: "The tumor is grossly oval, whitish, and measures 12 x 6 mm. Cross-section shows solid yellowish, translucent tissue surrounded by a thin fibrous whitish capsule. *Microscopically* the tumor of the cord is solid, the center is hyaline and does not show any nuclear stain. It is surrounded with hyaline fibrous tissue with a few slender fibroblasts. No epithelial structures are found in the tumor. *Diagnosis:* Fibroma of spermatic cord with hyaline degeneration (Figures 1 and 2)."

DISCUSSION

Of the almost 300 spermatic cord tumors reported, the majority are mesodermal in origin.[12] The presence of blood vessels, the vas deferens, connective

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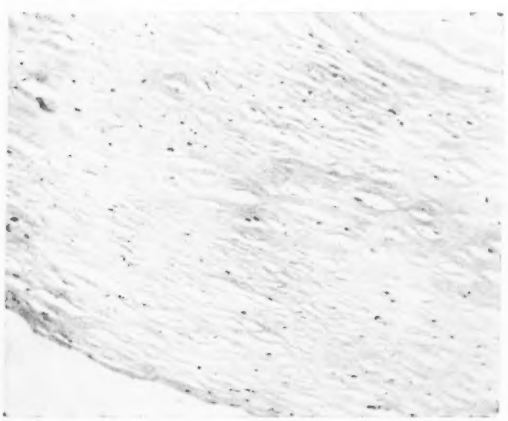

Figure 1. Cross section of the encapsulated tumor. Hyaline degeneration of the center with formation of clefts. In the periphery are coarse bundles of collagenous fibers with very few cells. (x 15)

Figure 2. Higher magnification of periphery of the tumor. The fibrocytes do not vary in size and there are no mitotic figures. (x 150)

tissue, nerve fibers, and fat in the spermatic cord may explain the complex constitution of so many of these tumors. As a rule it is not until the surgical specimen is examined under the microscope that an accurate diagnosis is possible.[17]

When malignancy does occur it is sarcomatous in 80 per cent of the cases.[14] Strong[12] feels that the malignant condition develops in previously benign tumor. The malignant tumors reported tend to be vicious in their behavior with widespread metastases and a very poor prognosis.

The ease of removal of most spermatic cord tumors strengthens the case for excision of all spermatic cord masses.

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The world has a way of giving what is demanded of it. If you are frightened and look for failure and poverty, you will get them, no matter how hard you may try to succeed. Lack of faith in yourself, in what life will do for you, cuts you off from the good things of the world. Expect victory and you make victory. Nowhere is this truer than in business life where bravery and faith bring both material and spiritual rewards. —*Preston Bradley*

PRESIDENT'S PAGE

DEAR DOCTOR:

Babson's says that our Capitol is confused over Ike's plans. This could be important to medicine. I think he will run. We have a more imminent problem, however, in H.R. 7225.

This bill, which so hastily passed the national legislative House, is now being heard in the Senate Finance Committee. Your president plans to appear before this committee to present the reasons Kansas doctors are not in favor of such a bill. You have read the recent brochure sent you concerning this dangerous threat to our nation. Now is the time to let your senator know how you and your friends feel about this legislation. Such Social Security amendments are a threat to good medical care. Also, these proposed changes might result in a drain of funds that could imperil the Social Security trust fund and critically weaken the nation's economy. A costly program of health and accident insurance should not be abruptly combined with the present system of providing pensions for the aged.

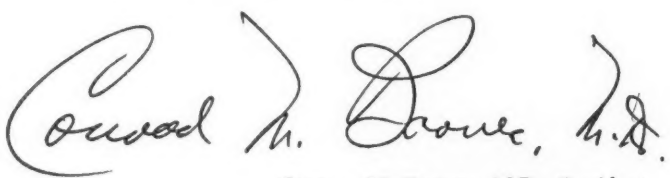
I fear that this H.R. 7225, which would give a disabled person his OASI pension at age 50 and lower the retirement age for women to age 62, is just a "foot-in-the-door" for a compulsory federal care program. By 1975 OASI taxes would probably be double the present amount. This is dangerous legislation!

Do not be complacent about H.R. 7225!

Dr. Cyril V. Black, chairman of our endowment committee and councilor, advises me that we are improving our status with the A.M.E.F. Dr. Black met with the national council of the A.M.E.F. in Chicago on January 22, and he reports that the University of Kansas received $38,981 in 1955. This amount exceeds the 1954 figure by about $9,500. Three hundred fifty doctors contributed in 1955, as compared to only 64 in 1954. This evidence of increased physician participation is greatly appreciated by me. Several of our counties had 100 per cent membership contributions. Thank you, doctor! But we still need more contributors and larger contributions. The doctors of Topeka, Kansas City, and Wichita need to get behind this program.

Be alert to do your part in the forthcoming medical assistants training program. See that your medical assistant attends this series of meetings that has been planned by our medical society committee, headed by Dr. Murray Eddy, and the Kansas Medical Assistants' Society.

Sincerely, in the practice of the Art,



CONRAD M. BARNES, M.D., *President*

EDITORIAL COMMENT

Our Centenarian Dr. George M. Gray

For the first time in its history the Kansas Medical Society now has an opportunity to salute a past president on the 100th anniversary of his birth. Dr. George M. Gray, Kansas City, will be 100 years old on Sunday, March 4, 1956.

Although not many people can celebrate such an event, that fact alone is not outstanding. What is

George M. Gray, M.D.

important is that Dr. Gray has not merely existed for 100 years; he has *lived* for 100 years, and he enjoys life today. He is alert, interested, progressive. He still looks forward.

To make the occasion a pleasant one for Dr. Gray, a number of friends are planning a dinner party in his honor. It will be held at the Town House Hotel, Kansas City, on Sunday, March 4. Reservations may be made through Dr. C. C. Nesselrode, 1200 Huron Building, Kansas City 10, Kansas.

Through his years of service to the Society, Dr. Gray made countless friends. Those who can be in Kansas City on the anniversary are urged to make reservations immediately so that the guest of honor can enjoy the celebration in the company of those who have a part in the Society and professional activities he loves to recall.

Although he was born in Waukegan, Illinois, March 4, 1856, Dr. Gray became a Kansan at the age of two when his parents moved to Wyandotte County. He attended public schools and Palmer's Academy in Kansas City and then studied medicine at the College of Physicians and Surgeons, Kansas City, Missouri. He was graduated in 1879 and spent the following year at Bellevue Hospital Medical College, New York.

Dr. Gray began practice in Kansas City in 1880 and has never formally retired. In 1913 he began limiting his practice to surgery. He became a member of the surgical staff of St. Margaret's Hospital in Kansas City when it opened its doors, and he served as chief of its surgical staff for more than 15 years.

His services to organized medicine are legion. He was willing to serve in any capacity, and his talents were frequently employed in various offices, including that of treasurer. In 1912 he held the highest professional office to which a physician in this state may aspire, president of the Kansas Medical Society.

Kansas City, too, benefited from Dr. Gray's guiding hand. He was interested in civic affairs and willingly served as mayor when a vacancy occurred in that office during a period in 1906 and 1907.

Medical Military Aide to the Governor was the title Dr. Gray carried from December 10, 1917, to January 4, 1919, when he was a member of the United States Medical Corps.

A busy life it has been, yes, a productive life, a life of service. Surely in reviewing the events of the past hundred years Dr. Gray can take satisfaction in the knowledge that he gave his best, that his achievements were many, and that his efforts are remembered and appreciated. Of his kind there can never be enough.

The best wishes of the Editorial Board and of the entire membership of the Society go to Dr. Gray at this time. It is the hope of all that March 4, 1956, will be a joyous occasion he will remember with pride as he starts on his second century of life.

Reducing the Budget

Editor's Note. At the time this* JOURNAL *reaches its readers, the Kansas legislature will have acted on its problem of finance. It will have reduced the state's budget or it will have provided for increased revenue through taxation. The following letter, sent to the editor of each newspaper in the state, was written when the legislature was considering a reduction of 5 per cent in each appropriation, a reduction which would be of great importance to the University of Kansas School of Medicine.

January 19, 1956

Dear Editor:

This open letter concerns a subject the doctors of Kansas believe to be important, and we would be grateful if you were willing to share it with your readers.

There are a few things even more important than

money, and one of these is health. It is strange that health isn't of very great concern until we become sick, and then we spare no effort or expense until we are well again. Often the treatment is much more costly than a little care in advance, which is to say that an ounce of prevention is worth a pound of cure.

That brings up the purpose of this letter. We are hearing rumors that an effort will be made to take a percentage from the budget of every state agency in an economy move. We are as interested in economy as is anyone, but we must remind ourselves that non-selective and careless budget cuts can be not only foolish but dangerous.

We are more familiar with health and therefore illustrate the above statement from examples out of that field. Our state operated schools are expensive, but reflect for a moment the cost of not operating these schools!

Visitors to the campus of Kansas State College at Manhattan will be shocked to see the condition of the facilities for student health services. However important any other item might appear, can the state afford to take less care of its greatest resource—its young people—than for its other property? But it is true at Kansas State College, and a thoughtless reduction in appropriations can only increase this serious problem and postpone its needed solution.

Then consider for a moment an opposite situation. At the University of Kansas School of Medicine you will see a beautiful facility, wonderfully equipped and rendering a most magnificent service to the people of Kansas in caring for the sick, in research, and in educating doctors of medicine.

This budget could be reduced, but where? Where could you take five per cent and retain the service that is now given? Suppose you take out five per cent of the hospital beds, or reduce by five per cent the number of students enrolled. This would be six in each class, six doctors less every year for Kansas communities that could never be made up in any other way.

Or take five per cent from hospital services—from nursing or laboratories, and let the patient take the risk. Or drop a subject from the curriculum and send the doctor out to practice without education in some one field. Or take it out of graduate education and let your personal physician keep up with medical advancements by going to schools in other states if he is to keep up at all.

Any or all of these things can be done to save money. If the appropriation to the University of Kansas School of Medicine is reduced, something like this will happen! And, of course, it will make no difference to you—unless you happen to be the patient who needs the services that have been taken away. Or unless your town would have received one of those doctors who did not get to study medicine at all. That would have made a difference. Or if your family physician had not attended the circuit course on exactly the subject of your illness because the school couldn't afford to give the course—that would have made a difference.

Yes, we are interested in economy but where the School of Medicine is concerned, please take our word for it because we know that the dollars spent here are ounces compared to the pounds it would cost to repair the loss.

Sincerely,
Conrad M. Barnes, M.D., President
The Kansas Medical Society

Science Fairs

Have you seen the thrill of discovery on the face of a young scientist as he sees an ameba for the first time in his life? Or have you witnessed the breathless anticipation of the youngster digging for fossils in a limestone rock, or the achievement in his mind as he views the rings of Saturn for the first time, or the world that opens for him upon his introduction to trigonometry?

Are his discoveries any less real or less significant because they are new only to him? Even though the seas have been charted by previous explorers, his first journey is as vivid and as exciting as though it had never been made before. Then guide this young boy or girl for only a short time. Teach him to anticipate an expanding horizon, to look forward to never ending discoveries, and soon you will have laid the foundation for a career in science.

The idea of a Science Fair is to do exactly that—to inspire boys and girls to search into the unknown, to give them the pleasures of discovery, to create an interest in a scientific career. This may be in any of the sciences, medicine as truly as physics or astronomy, or mathematics.

Whether it will be in the field of medicine depends upon the direction or guidance that comes from physicians. And up to now that has been small!

There were last year 71 state or regional science fairs in which some 350,000 youngsters competed directly or indirectly. Here were displayed original achievements in research or in construction or in accumulation of data. These children were guided through some 15,000 science clubs in high schools, or by adults who were trying to interest them to become physicists, astronomers, chemists, nurses, or active in any of a hundred other scientific fields.

And some were exploring the field of medical science, but not because organized medicine was alert

to its opportunities for only three of the 71 fairs were sponsored by medical societies, two in Indiana and one in the District of Columbia.

In Kansas there will be three science fairs in 1956. These will be regional events with most of the geographical area of Kansas included. Boys and girls in hundreds of high schools are now working on problems to exhibit. They range from an attempt to analyze the color in butterfly wings to a methodical exploration of pi. Included are principles of genetics applied to the creation of hybrid corn, and the chemical evaluation of the contents of Kansas River water, a fabric tension testing experiment, and an original aircraft design, and many more.

But neither the Kansas Medical Society nor any county medical society in this state has at this time taken a single step to aid these young researchers in their medical experiments. Organized medicine has not tried to stimulate students into studying medicine, and neither have societies utilized this most wonderful opportunity for showing these future scientists what medical research means and what it means to them.

Actually it is not too late. Any individual who believes in young people may approach the science club of the high school in his town and offer to help. Any society may still make this a project for 1956 and have a committee perform this most vital service to youth and at the same time benefit by a beautiful public relations gesture. It is hoped that many may respond in this enterprise—now.

Mid-West Cancer Conference in March

Plans are rapidly being completed for the eighth annual Mid-West Cancer Conference, to be held at the Broadview Hotel, Wichita, March 22 and 23. The Committee on Control of Cancer of the Kansas Medical Society and the Kansas Division, American Cancer Society, are joint sponsors of the event.

In general, the format of previous conferences will be followed. Scientific programs will be presented each morning and afternoon. A banquet on Thursday evening, March 22, will be open to all physicians attending the conference and their wives.

Each speaker who will take part in the program is a specialist widely known throughout the nation. Complete programs will be sent to all members of the Society by direct mail. Biographical sketches of four of the speakers are presented below, and information about others will be published in the March issue of the JOURNAL.

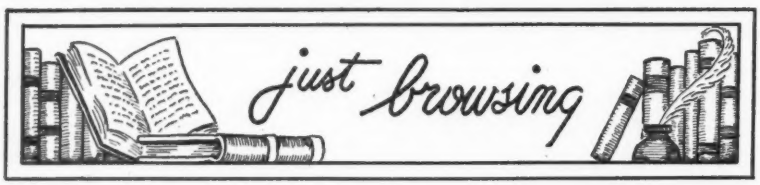
One of the participants, William Ogburn Russell, M.D., is pathologist-in-chief at the M.D. Anderson Hospital and Tumor Institute, Houston. He is also professor of pathology at the Postgraduate School of Medicine of the University of Texas and associate professor of pathology at Baylor University College of Medicine. He received his degree from Stanford Medical School in 1938. In addition to being a founding fellow of the College of American Pathologists, he is a diplomate of the American Board of Pathology and a member of the American Society of Clinical Pathologists, the American Society of Experimental Pathology, and the International Academy of Pathology.

A second speaker is George D. Snell, Sc.D., who has been research associate for the Roscoe B. Jackson Memorial Laboratory, Bar Harbor, since 1934. His graduate work for the Sc.D. degree, completed at Harvard in 1930, was in the field of mammalian genetics. As a National Research Council fellow at the University of Texas, 1931-1933, he initiated a study continued at the Jackson Laboratory on induction of x-rays and neutron rays of hereditary changes in mice. He is now studying factors determining susceptibility and resistance to tumor homografts.

A 1926 graduate of the University of Kansas School of Medicine who will take part in the program is William J. Engle, M.D., urologist at the Cleveland Clinic Foundation. He has won many professional honors and is now serving as president of the Cleveland Academy of Medicine. He is a diplomate of the American Board of Urology, a fellow of the American College of Surgeons, and a member of the American Urological Association, the American Association of Genito-Urinary Surgeons, the Clinical Society of Genito-Urinary Surgeons, and the International Society of Urology.

Bentley Prescott Colcock, M.D., another guest speaker, has been a member of the staff of the Lahey Clinic, Boston, since 1939. He received his medical degree from the University of Pennsylvania in 1933 and was a fellow in surgery at the Lahey Clinic from 1936 through 1938. He served as a lieutenant colonel in the Army Medical Corps during World War II. Dr. Colcock is a diplomate of the American Board of Surgery, a fellow of the American College of Surgeons, and a member of the New England Surgical Society, the Boston Surgical Society, and Excelsior Surgical Society.

Interest in the Mid-West Cancer Conference has been continuing to grow through the years. Registrants from states other than Kansas always attend, and each year sees an increase in the number of in-state physicians present. Hotel reservations should be made immediately.



Though it may not be wise to mention it openly in a medical publication, we have felt for some time that modern medical writing suffers from an excessively objective and scientific approach. The cold pronouncement of medical fact, case study, or experimentation permits little or nothing of the author's personality to appear in the opus.

After the perusal of a couple of old volumes, we suggest it may be well to return to the earlier custom of "presenting" the material to some individual of intellectual or financial standing by means of a dedicatory preface. We present herewith some examples which may be considered crudely obvious by today's standards but seem to have served well in their time.

One of these is the dedication by Percivall Pott of his *Practical Remarks on the Hydrocele* to one Peter Sainthill, Esq., in 1762. He says, "Some time ago I gave you the trouble of looking over the following sheets in manuscript, with which you said you was much pleased.

"The approbation of so good a judge has emboldned me to print them, and I do not know a more proper person to whom to address them.

"The rank you have long and deservedly borne in the profession and the character you have always maintained in it as an honest man, as a judicious and fair practitioner, give you a just claim to the regard of all its members.

"By this address I mean to express my sense of your merit, and my estimation of your friendship; but lest what I intend as a civility should become offensive, I will not add anything which though strictly true might have the appearance of flattery, nor make a needless recital of those good Qualities which you are so well known to possess.

"I heartily wish you continuance of health, and am, Dear Sir, Your most obedient Servant,
Percivall Pott."

Another medical gentleman who turned a neat phrase of praise was William Harvey whose *De Motu Cordis* was published in 1628 and dedicated to King Charles in this manner:

"To the Most Illustrious and Invincible Monarch Charls King of Great Britain, France, and Ireland, Defender of the Faith. Most Gracious King, The Heart of creatures is the foundation of life, the Prince of all, the Sun of their Microcosm, on which all vegetation does depend, from whence all vigor and strength does flow. Likewise the King is the foundation of his Kingdoms, and the Sun of his Microcosm, the Heart of his Commonwealth, from whence all power and mercy proceeds. I was so bold to offer to your Majesty those things which are written concerning the Heart, so much the rather, because (according to the custom of this age) all things human are according to the pattern of man, and most things in a King according to that of the Heart. . . . You may at least, best of Kings, being plac'd in the top of human things, at the same time contemplate the Principle of Man's Body, and the Image of your Kingly power. I therefore most humbly intreat, most gracious King, accept, according to your accustom'd bounty and clemency, these new things concerning the Heart, who are the new light of this age, and indeed the whole Heart of it, a Prince abounding in vertue and grace, to whom we acknowledge our thanks to be due, for any good that England receives, or any pleasure that our life enjoyes:

Your Sacred Majesties most devoted Servant,
William Harvey."

He addresses the same volume to another colleague so: "To the Most Excellent and Most Ornate man, D. ARGENT, President of the College of Physicians in London."

Some of our contemporary brethren fulfill the excellent part, but the ornate are harder to find. Nevertheless, it is suggested that aspiring contributors to this journal may well consider opening their future offerings in some such fashion:

"To the Most Wise and Gorgeous Hunke of Edytor, Orville R. Clark."

On second thought, perhaps not.—*D.E.G.*

Clinicopathological Conference

The Differential Diagnosis of Nephrosis

CASE PRESENTATION

Today we are considering the case of a 45-year-old white female who was admitted to the University of Kansas Medical Center on March 8, 1955, complaining of progressive swelling of her entire body since August 1954. She expired on the evening of April 4, 1955.

The patient was apparently in good health until August 1954 when she had an illness characterized by fever and malaise of six or seven days duration which she called "flu." She felt well for about three weeks afterward, until she noted ankle edema for the first time. Her physician told her that she had albumin in the urine and prescribed a low protein diet and some medication. The swelling progressed, however, and the patient experienced increasing malaise and fatigue.

In November 1954 she was hospitalized and put on a high protein, low salt diet. This was not effective in decreasing the edema. She was given 80 units of corticotropin intramuscularly daily for 10 days, but the anasarca only increased. In January 1955 she was given 100 grams of salt poor albumin intravenously daily for five days, and she had marked diuresis and lost 20 pounds. Her malaise persisted, however, and the edema returned despite salt restriction, diuretics, and ion exchange resins.

The only past illnesses of note were two periods of hospitalization in 1951 and 1953, when the patient was given electroshock treatments for mental depression.

The patient had one brother who died in diabetic coma at age 25 and one sister who was a diabetic.

The only feature of interest in the review of systems was an intermittent diarrhea of two to three loose, liquid stools a day.

When admitted to KUMC the patient appeared to be a well-developed, well-nourished white female who was chronically ill. The outstanding feature of the examination was generalized edema and ascites. The blood pressure was 90/60; the pulse was 88 and regular. There were numerous petechiae on her face and other areas of her body. The eye-grounds were normal. The tongue was red and beefy in appearance. There were scattered moist rales in the lung bases. The heart sounds were poorly heard, but a definite gallop rhythm was present. The heart size was difficult to ascertain. There were no palpable abdominal masses, but massive anasarca was present. There was edema of the skin of all areas of the body.

Cursory inspection of this clinical history, the physical findings, and the laboratory data quickly suggests a nephrosis, but little more that is specific. The persistent diarrhea, the absence of hypertension, the presence of a gallop rhythm, petechiae, and non-specific electrocardiographic changes were items suggesting some unusual disease entity with widespread visceral damage. No single laboratory procedure, other than a biopsy, had any helpful potential. While the true character of amyloid still eludes us, new and interesting light is being given to the clinical problem with the discovery of a familial characteristic in primary amyloidosis.

A heavy precipitate of albumin was found in the urine. The specific gravity was 1.006, and the specimen was loaded with clumps of pus cells and some hyaline and granular casts. There was no glycosuria. The red count was 5,400,000 with a hemoglobin of 15.0 grams. The white count was 16,000 with 74 per cent polys (57 filamented and 17 non-filamented), 14 lymphocytes, 2 eosinophiles, and 10 monocytes. The platelet count was 220,000. Serology was negative. The serum calcium was 4.0 milliequivalents per liter; phosphorus, 3.7 mEq/L; sodium, 127 mEq/L; potassium, 5.3 mEq/L; CO_2, 17.8 mEq/L; chloride, 98 mEq/L; non-protein nitrogen, 43 milligrams per 100 ml.; creatinine, 2.5 mg. per 100 ml.; sugar, 68 mg. per 100 ml. There was 9.75 per cent excretion of phenolsulfonphthalein in one hour, and the urea clearance was 11 per cent of normal. No Bence-Jones protein was found in the urine. The Addis count was 209,000,000 white cells, 1,760,000 red cells, and no casts in a 24-hour urine specimen. There were 6.9 grams of protein in an 800 ml. 24-hour urine specimen. Urine cultures showed a heavy growth of

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Edited by Jesse D. Rising, M.D., and Mahlon Delp, M.D., from recordings of the conference participated in by the departments of medicine, surgery, radiology, and pathology of the University of Kansas Medical Center as well as by the third and fourth year classes of medical students.

E. coli. Blood cultures were negative at 10 days. The total serum bilirubin was 0.2 mg. per 100 ml.; serum cholesterol, 984 mg. per 100 ml. with 72 per cent esters; serum albumin, 1.49 grams; serum globulin, 2.31 grams; alkaline phosphatase, 1.9 millimols; cephalin cholesterol flocculation, 1 plus; thymol turbidity, 42 units. I_{131} uptake was 13 per cent. No LE cells were found in the blood. There was 30 per cent retention of Congo red.

On admission the patient was placed on a low sodium diet. She soon experienced nausea, which persisted through the remainder of her hospitalization and severely limited her food and fluid intake. On March 13 she was given one unit of whole blood. From March 17 to March 24 she received a total of 450 grams of salt poor human albumin intravenously in daily divided doses. Her weight did not change, and there was no diuresis. The daily urine output remained at 1000 to 1500 ml. from admission until March 23, after which it never exceeded 500 ml. The serum sodium remained between 120 and 125 mEq/L, and the serum chloride remained below 90 mEq/L during the greater part of her stay. Before March 28 the serum CO_2 was between 15 and 20 mEq/L, and thereafter it was between 8.0 and 15 mEq/L.

On the fourth day after the salt poor albumin was started, the total proteins rose to 4.5 grams per 100 ml. with 3.56 grams of albumin and 0.94 grams of globulin. At this time the cholesterol fell to 310 mg. per 100 ml., and on March 31 it dropped to 190 mg. The blood proteins were essentially unchanged during the remainder of her life, even after salt poor albumin was stopped. At all times the urine sodium levels were above 50 mEq/L and chloride over 30 mEq/L.

On March 25, the third day after her oliguria had become manifest, the patient was given 10 units of corticotropin by slow intravenous drip, and this was repeated daily for 10 days. During this time her condition continued to deteriorate with increasing anasarca, oliguria, malaise, nausea and anorexia, and extension of the petechial rash on various parts of her body.

During the last four days of the patient's life, the daily urine output was between 50 and 200 ml. On March 21 the non-protein nitrogen was 51 mg. per 100 ml., rising to 88 mg. on March 30 and to 142 mg. on the day of death. Attempts were made to promote diuresis by the intravenous administration of hypertonic sodium chloride, but this was without effect. Southey tubes were inserted in her legs and thighs on March 31, and about five liters of fluid were drained in 72 hours. She became progressively weaker, more dyspneic and somewhat irrational, and died quietly on April 4, 1955.

Dr. Mahlon Delp: One item that wasn't emphasized in the protocol was that this patient had a diarrhea which, I believe, began in November at the time she was first hospitalized and continued until her admission here. It appears that diarrhea must not have been much of a problem during her stay here. Do you recall the diarrhea, Dr. Allen?

Dr. Max Allen (internist): She did have an intermittent diarrhea, but it was no problem during her stay here.

Dr. Delp: Are there any questions for Dr. Cochran?

Mr. Ernie Chaney (senior medical student): Did she have any dysarthria or dysphagia?

Dr. T. E. Cochran (resident in medicine): The tongue was red, beefy, and swollen, and she did have some difficulty in swallowing.

Mr. Chaney: Was a prothrombin time done?

Dr. Cochran: Her admission prothrombin time here was 97 per cent of normal.

Question: Was the patient digitalized?

Dr. Cochran: Not at this hospital.

Mr. Terry Denison (senior medical student): What were the results of the skin tests?

Dr. Delp: I don't think there are any skin tests recorded on this patient.

Mr. Jess Charles (senior medical student): I should like to know whether she showed hypertension at any time.

Dr. Cochran: The blood pressures were low throughout this stay.

Question: Was there pus in the urine?

Dr. Cochran: Yes, there was. Various urinalyses during her first week in the hospital were also reported as being loaded with red cells, and this continued until her terminal phase.

Question: Were electrolytes taken on the day of death?

Dr. Delp: The non-protein nitrogen on the day of death was 142.5 mg. per 100 ml.; creatinine, 6.4 mg.; sodium, 122 mEq/L; potassium, 5.4 mEq; CO_2, 8.8 mEq; and chloride 88 mEq.

Question: What were the results of serology studies?

Dr. Cochran: They were negative.

Mr. Chaney: Did she have any muscular twitching or convulsions terminally?

Dr. Allen: There were none at any time.

Question: Were petechiae ever noted while taking the blood pressure?

Dr. Cochran: Not to my knowledge.

Dr. T. K. Lin (cardiologist): What was the temperature course in the hospital?

Dr. Delp: There was no elevation above 98.6.

Question: Was a muscle biopsy taken at any time?

Dr. Cochran: There was no biopsy of any kind.

Dr. Delp: Mr. Ashley, will you show us the electrocardiogram.

Mr. John Ashley (senior medical student): This tracing (Figure 1) was made on the second hospital day. The rate is approximately 80, and there is a normal sinus rhythm. Low voltage is apparent throughout, with T wave flattening in all leads. Subsequent tracings show virtually the same thing.

Dr. Delp: Is there anything there that is specific or would help in your differential diagnosis, Mr. Ashley?

Mr. Ashley: Possibly the generalized low voltage.

Dr. Delp: Do you have any comments about these tracings, Dr. Lin?

Dr. Lin: There is a low voltage and there are nonspecific T wave changes.

Mr. Kendall Davidson (senior medical student): This first x-ray, a P-A view of the chest (Figure 2), was taken on the day following admission. Within the bony thorax I see no abnormalities or soft tissue changes about the thorax. In the lung fields themselves there is a diffuse haziness bilaterally, more pronounced in the bases. This picture is consistent with diffuse inflammation and possibly fluid in the pleural spaces. There is also a widening of the heart at the base. This may be chamber enlargement, dilatation, or pericardial effusion. The lateral view taken at the same time shows diffuse haziness over the middle and lower lung fields. The cardiophrenic angle is obscured, suggesting either fluid or cardiomegaly.

Dr. Delp: Dr. Tice, do you have any comment on these films?

Dr. Galen Tice (radiologist): The observations noted are entirely correct. There is blunting of the costophrenic angles and possibly fluid. I think his observations about the heart contour were good. It had a water bottle contour.

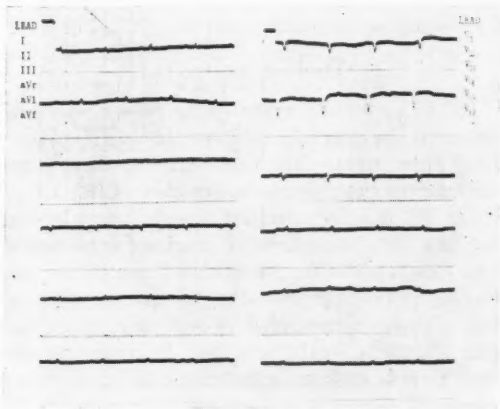

Figure 1

Figure 2

DIFFERENTIAL DIAGNOSIS

Dr. Delp: Mr. Chaney, I would like to have your summary of the important items in the protocol and your differential diagnosis.

Mr. Chaney: The case today is that of a 45-year old white woman who was apparently in good health until eight months prior to her death, at which time she suffered an upper respiratory infection with fever and malaise. Three weeks after this there was the onset of pedal edema and albuminuria. Her edema increased, and she was hospitalized and given salt poor albumin, corticotropin, and a low salt diet in an attempt to induce diuresis. Diuresis occurred but her edema reappeared, and she was admitted to this hospital.

Upon admission here she was found to have what was clinically a nephrotic syndrome with hypercholesterolemia, hypoproteinemia, and proteinuria. She had petechiae and a gallop rhythm. She progressed in this despite treatment with salt poor albumin, low salt diet and, terminally, hypertonic salt solution.

The important laboratory findings are in the protocol, so I won't repeat them. Another important thing, as Dr. Delp mentioned, was the diarrhea which was not adequately explained.

I will base the differential diagnosis today on causes of the nephrotic syndrome. First of all, we should consider toxic nephrosis which may occur with the ingestion of drugs, infections, or the toxemia of pregnancy. Because of a negative history in this case, I shall rule these out. The next thing I would like to consider is that sometimes syphilis can involve the kidney, causing a nephrotic syndrome. This patient had negative serology and no history of lues, so I shall rule out syphilis in this case. Diabetic nephropathy may result in a nephrotic syndrome; however, such patients usually have hypertension, retinal changes, and often a family history of diabetes. She had no clinical or laboratory history of diabetes. Disseminated lupus erythematosis may result in a nephrotic syndrome, but this patient did not have arthralgic pain which is one of the manifestations of the disease, and she had a negative LE cell preparation. Pure lipoid nephrosis can give this picture; however, it usually occurs in children, with eventual recovery.

Now I come to three diseases which we have to consider a little more seriously. These are the amyloid diseases of the kidney which are usually classified in three groups: primary amyloid disease, secondary amyloidosis, and tertiary amyloid disease as seen in multiple myeloma.

This patient was a female; she had no bone pain, no x-ray evidence of multiple myeloma, and no Bence-Jones protein. So I think I can rule out multiple myeloma.

Secondary amyloidosis usually comes from a chronic debilitating disease. It has been described in tuberculosis, bronchiectasis and ulcerative colitis. She had none of these. She had a negative Congo red test. It is considered to be positive if 80 to 90 per cent of the dye is retained, but only 50 per cent of patients with secondary amyloidosis have a positive Congo red test. Amyloid usually involves organs such as kidney, liver, and spleen below the diaphragm. Even though she may have had involvement of her liver and spleen, they could not be felt. On this basis I rule out secondary amyloid disease.

Primary amyloidosis is a rare disease. It usually involves the small vessels of the skin, tongue, and myocardium. In some 20 per cent of the cases it involves the kidney. This patient had a number of things which make me think of primary amyloid disease. She had hypotension, hypercholesterolemia, diarrhea, petechiae, and the nephrotic syndrome. The fact that she had a negative Congo red test does not disturb me, because primary amyloid picks up the dye less than does the secondary.

The next lesion to consider is bilateral renal vein thrombosis. This occurs rarely. There were five cases in adults reported in the literature up to December 1954. It occurs more commonly in children, typically in a child who has diarrhea and who is dehydrated, with an onset of acute flank pain and hematuria. They usually have a lateral palpable abdominal mass, and the clinical course is usually hopeless. Because of the rarity of the disease and because of the clinical picture, I rule out bilateral renal vein thrombosis.

Another condition to consider is subacute or chronic glomerulonephritis. These two entities are difficult to differentiate clinically and pathologically. They can result in the nephrotic syndrome. The usual course is longer than in this case. Terminally these patients may have hypertension. I lean toward that diagnosis, but, in summary, I would say that I cannot entirely rule out renal vein thrombosis and primary amyloidosis.

As for the patient's hospital course, approximately four days prior to her death she went into an oliguric state. This is difficult to evaluate. I thought that probably she was in a low salt syndrome when she was admitted to this hospital, and that she continued in this condition. The plasma volume of patients with the nephrotic syndrome has been shown to be some 20 to 30 per cent lower than in the normal individual. These two factors together may have resulted in a decreased blood flow to the kidneys which were already damaged, and some type of tubular lesion akin to lower nephron nephrosis occurred, and the patient went on into uremia.

We also have to consider the possibility that her terminal event was one of hyperpotassemia, and that she received a hypertonic saline solution on the day of death and may have had a cardiovascular collapse due to overloading of the cardiovascular system.

Dr. Delp: Thank you, Mr. Chaney. I choose to think that you included the consideration of bilateral renal vein thrombosis out of respect to me. I have to admit that that was my diagnosis of this patient, and I publicized it too widely.

CLINICAL DISCUSSION

Dr. Delp: I really wish to hold the major discussion after the pathology is presented, but I can't refrain from asking Dr. Kaul and Dr. Berry what their differential diagnosis might have included.

Dr. Phil Kaul (internist): I might begin by saying that the student gave an excellent presentation. One must consider the possibility of constrictive pericarditis in any nephrotic-like syndrome, or anasarca. The lack of hypertension at any time during the patient's course would be against the nephrotic syndrome as a result of glomerulonephritis. It makes one think more in terms of nephrosis, possibly due to amyloid disease.

Dr. Max Berry (internist): First of all I want to say that was an excellent presentation, and I am sorry to say that I don't have my thoughts as well organized as the student did.

This woman probably had a nephrotic syndrome, whatever the nephrotic syndrome is, and, in addition to that, one has to consider what complication might have occurred to cause her death. I don't know, and am a little bit reluctant to ask, through innate modesty, whether this woman had any cortisone. Aggravation of the glomerular lesion would make me hesitant to give any cortisone. I would think that corticotropin would do the same thing. I don't know that I would have handled the patient any differently. The possibility of adrenal cortical atrophy must be considered because this woman had certain things to suggest it, such as low blood pressure and diarrhea, and the fact that she deteriorated progressively in spite of what I think was good medical treatment. If she had enough cortisone to suppress the function of her adrenal cortex, it is a possibility that she died in adrenal cortical insufficiency.

Dr. Delp: Mr. Buie, what was your diagnosis?

Mr. Dan Buie (senior medical student): Primary amyloidosis.

Dr. Delp: It was said in the protocol that this patient had a febrile illness as the first evidence of sickness. She had little thereafter. How do you explain that?

Mr. Buie: I do not think that was part of this illness. If I were trying to make it part of the illness, I would think of acute glomerulonephritis, but I don't think we have evidence for that diagnosis.

Dr. Delp: How do you do a Congo red test, Mr. Cox?

Mr. Wallace Cox (senior medical student): A milliliter of 1 per cent Congo red solution is injected intravenously, and the blood concentration is determined at intervals, including a one-hour determination. If less than 40 per cent of the dye is absorbed, the test is negative. If over 40 per cent, and especially if over 90 per cent of the dye is absorbed, the test is considered indicative of amyloid disease.

Dr. Delp: Mr. Denison, is this a normal Addis count?

Mr. Denison: No. There is elevation in the Addis count, particularly in the white and red cell count. The normal white cell is six-tenths of a million, and the patient had 209,000,000. The normal red count is approximately one-tenth million.

Dr. Delp: Is the Addis count a procedure of any merit?

Mr. Denison: I question whether it is of any real merit in this case. The increase in white count is evidence of infection in the kidneys, such as pyelonephritis, but it is a common observation in patients with the nephrotic syndrome. The elevation of the red count indicates that she was losing red blood cells. I don't know why, unless it was due to breakdown of the glomerular integrity. This is possible in a severely damaged kidney like she had. I question the complete lack of casts in the Addis count, because a normal individual has a range of a few thousand casts. It is probably a mistake, particularly considering the amount of protein that is present.

Dr. Delp: Mr. Davidson, this patient had a thymol turbidity of 42 units reported on the liver function profile. Do you have any comments?

Mr. Davidson: In cases in which there is high cholesterol at the time of determination, the thymol turbidity may be unreliable.

Dr. Delp: Mr. Ashley, this patient's most outstanding evidence of disease must have been her edema. What do you think was the most significant physical finding that she had?

Mr. Ashley: I think that the presence of petechiae is outstanding.

Dr. Delp: You think this should have been the clue to the diagnosis, Mr. Chaney?

Mr. Chaney: I think the cardiovascular symptoms and the gallop rhythm.

Dr. Delp: Gallop rhythm! This certainly could be a most significant finding.

PATHOLOGY REPORT

Dr. Harlan Firminger (pathologist): This patient was about 65 inches in height and weighed about 170 pounds. She appeared reasonably well nourished and not debilitated. The abdomen was distended, and the skin everywhere was extremely pale except for the numerous petechiae. These petechiae were concentrated particularly in areas of motion, such as the eyelids, the angles of the buccal mucosa, the anterior axillary folds, and the inguinal folds.

About 4.5 liters of serous fluid was found in the peritoneal cavity. The thoracic cavity contained about 1200 ml. and the pericardial cavity about 250 ml. of similar serous fluid. All the organs were somewhat enlarged, due partly to edema. In many organs there were small, pinkish deposits which stained a deep mahogany brown with iodine. The picture was that of primary amyloidosis, and I shall present the organs in the order that they are most frequently involved by this disease.

The heart was enlarged and weighed 580 grams. The myocardium of both ventricles was thickened, and the chambers were somewhat dilated. There were tiny, dark, grayish-pink oval areas about 1 to 2 mm. in diameter throughout the myocardium. Plaques of similar material were present on the endocardium,

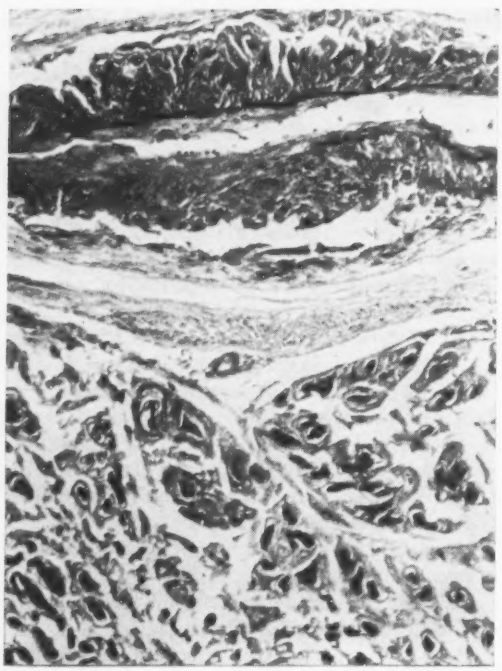

Figure 3. Photomicrograph of the myocardium demonstrating rings of amyloid around individual myocardial fibers. The wall of an adjacent coronary artery also shows extensive deposition of amyloid in the walls (crystal violet stain).

and vesicular appearing little pink nodules were present on every valve in the heart. All of these lesions gave a positive reaction for amyloid using iodine.

Microscopic sections from such an area in the endocardium revealed a large plaque of relatively homogenous eosinophilic material. In sections of the myocardium stained with hematoxylin and eosin, one sees an immense amount of eosinophilic deposit interstitially, surrounding many of the myocardial fibers and around the blood vessels. This ringing of the myocardial fibers is characteristic of amyloidosis. The crystal violet stain, which stains amyloid a bright red in contrast to nonamyloid tissues which are purple or blue, demonstrates a massive replacement in many areas of the myocardium by amyloid. A section of myocardium shows rings of amyloid about myocardial fibers and a large coronary vessel involved with amyloid (Figure 3).

The gastrointestinal tract is the next most frequently involved by primary amyloidosis and particularly the tongue. Unfortunately we do not have sections of the patient's tongue. We could see that the stomach was involved grossly, but it is not easily seen in the fixed specimen. In addition, there are numerous hemorrhages in the gastric mucosa. The duodenum shows tremendous deposits of eosinophilic material within the mucosa. The vessels are also involved. This is rather characteristic. The appendix shows tremendous amounts of the same material in the muscle and the subserosa.

The lung shows amyloid in the alveolar walls and around small blood vessels.

The kidneys are quite large and together weigh about 550 grams (Figure 4). They are pale with prominent red medullae, and the cortex is much thickened. The consistency is waxy. The architecture is obscure. There are petechiae in the pelves of the kidneys. Arteriosclerosis is not particularly striking either in the kidneys or elsewhere. A histologic section with hematoxylin and eosin stain shows the amyloid material around the capillaries and in the wall of the entering arteriole very brilliantly (Figure 5).

The adrenal gland shows a mass of amyloid material involving the entering vessels. Another section shows the amyloid even better extracellularly, more like that seen in secondary amyloidosis.

The liver has tremendous amyloid deposits around the blood vessels of the portal veins, but there is a lack of amyloid deposition along the sinusoids, such as one sees in secondary amyloidosis.

Small vessels of the pancreas are involved, and in some instances their lumens are narrowed. Generally the vessels are not particularly narrowed.

In the brain there are deposits which look like amyloid around the cerebral vessels, but we have not

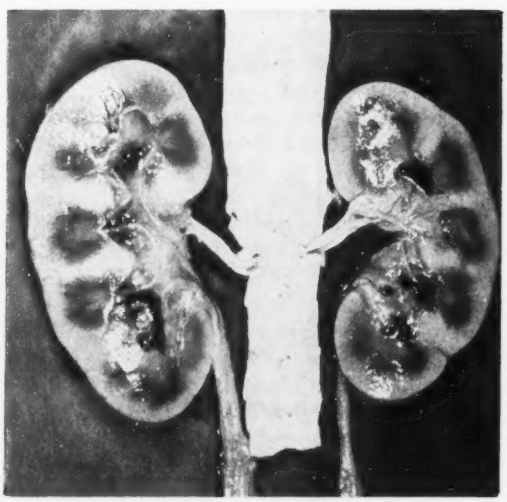

Figure 4. Gross photograph of the kidneys which are enlarged, pale, and firm with a waxy cut surface and obscure cortical architecture.

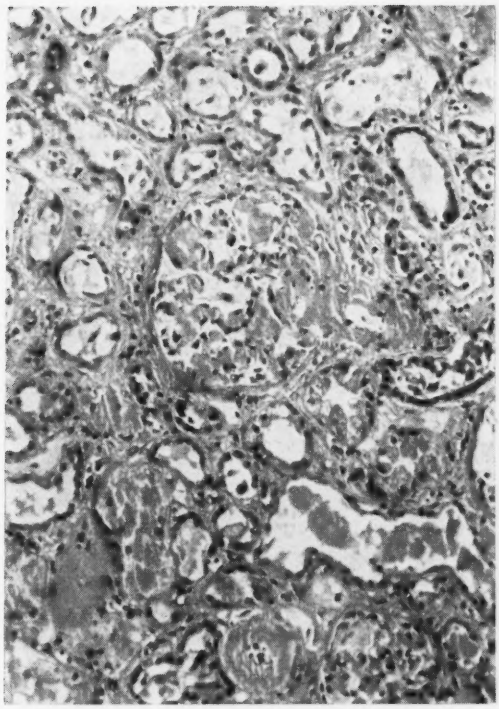

Figure 5. Photomicrograph of kidney showing deposition of amyloid in the glomerular tuft and entering arteriole. All the renal tubules are dilated and contain precipitated proteinaceous material. There is degeneration of the renal tubular epithelium (hematoxylin and eosin stain).

done a differential stain. The deposits do not involve the media to any extent but occur in the outer portion of the vessels.

Finally, the uterus is described as frequently being involved. It is a tremendous organ, which is soft, red, and boggy. Histologically it has amyloid deposits in the subserosal vessels. The crystal violet stain shows tremendous replacement of uterine muscle by bright red staining amyloid material.

Now, this would appear to add up nicely to a fine case of primary amyloidosis, with all the essential findings. The types have already been described by the student, and I will not reiterate them. I would like to mention a little about the clinical pathological correlation.

This severe, apparent pallor of the patient was due to amyloidosis of the skin. The cutaneous vessels and the dermis were involved, and the skin was diffusely edematous. Hypotension is characteristic of this disease in contrast to acute glomerulonephritis with which this might be confused. A normal red blood cell count is uncommon in chronic nephritis. The petechiae occur characteristically in the areas subjected to motion or trauma, being due to amyloid involvement of the walls of the vessels in those areas in the skin. The rales in the lungs could be caused by edema or heart failure. The electrocardiographic changes are apparent, and these patients may have dysrhythmias caused by amyloid deposits. The urinalysis is characteristic, albumin and cells go through the involved glomeruli. There are tremendous numbers of casts in this case. I agree with the student that there was an error in the Addis count. The loss of sodium and chloride would apparently be renal. I think she died in uremia.

I did not mention the spleen, by the way; it was firm, and, in addition to extensive amyloid deposition, there was a healed infarct in it which is frequently found in this disease.

I would like to conclude by mentioning the theories which are suggested for primary amyloidosis; it is obvious that we do not know the cause. It may be a generalized disturbance in protein metabolism. It may be the result of an antigen-antibody reaction. Some have suggested that it belongs among the collagen diseases. My own interpretation at the moment is that it is an unexplained phenomenon which usually involves the smaller blood vessels and sometimes the larger, with perhaps a loss of albumin into and through the walls of peripheral vessels, much the same as occurs in the glomeruli. This causes edema of the organs, including subcutaneous tissues, and produces a reversal of the albumin/globulin ratio which is characteristic as in this case. Unfortunately, careful chemical analysis of amyloid still needs to be done so that we may have more definite information as to just what it is.

Mr. Chaney: What about the kidney tubules?

Dr. Firminger: The tubules were degenerated on the basis of casts within them and pressure atrophy of the tubules, and also perhaps some ischemia due to impairment of the flow of blood supplying them. There was some fat within them, but this could not be classified as a lipoid nephrosis. It was primarily a glomerular lesion with the tubular lesion being secondary.

One might ask why hypertension does not occur in these cases. Certainly the renal component is present. One would think that there would be decreased blood flow through these glomeruli. My suggestion is that the involvement of the heart may play some part in the lack of hypertension, but more important is the fact that the vessels throughout the body have their muscularis replaced with amyloid and cannot produce the increased peripheral resistance to give hypertension.

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Dr. Delp: What were the bone marrow findings?

Dr. Firminger: The bone marrow, if anything, was a little on the hyperplastic side. The femoral marrow was deep red, otherwise not peculiar.

Dr. Sloan Wilson (hematologist): No plasma cells?

Dr. Firminger: No plasma cells.

Dr. Albert Jackson (internist): Would a liver biopsy have given the diagnosis?

Dr. Firminger: A liver biopsy would not have helped unless you were lucky enough to get one of the big vessels. The usual method of diagnosing primary amyloidosis is by biopsy of the tongue.

Dr. Delp: Dr. Christianson has had considerable interest in this entity and has studied at least one case carefully. He has had contact with two other patients within the past year. I would like to ask for his comments.

Dr. John F. Christianson (internist): Certainly amyloid disease is a unique entity, particularly because of the abnormalities in the protein metabolism. It may be related to some of the lipoid abnormalities, such as the reticulum cell with deposition of abnormal protein-chondroitin-sulfuric acid material, which some workers think is identical with amyloid.

For some time we have thought that we could divide amyloid into at least three and possibly four groups: the so-called primary, or paramyloid; the secondary, or true amyloid, which follows granulomas or suppuration, tuberculosis, osteomyelitis, etc.; the amyloid which is found in close association with multiple myeloma; and the tumor forming or plasmacytoma. The true, or secondary type of amyloid, is generally deposited in the parenchymatous glands such as the liver, kidneys, spleen, and adrenals, whereas the primary type is generally found in the tissues of mesenchymal origin as Dr. Firminger outlined.

This case falls into the class of primary amyloidosis. It is my belief, however, that the thinking on amyloid disease is in for a revision, particularly in view of some work that is being done at the University of Michigan.

Ostertag,[4] in 1950, was the first to refer to a familial tendency in amyloidosis. Andrade,[1] from Portugal, published in 1952 the first convincing evidence of the familial nature of the disease. He studied 12 families, and, in two of them, was able to see patients of two generations.

In June 1953, Kantarjian and De Jong[3] of the University of Michigan published reports of three cases, a man and his two daughters, who died of proved primary amyloid disease. The three had identical courses with diarrhea, visual difficulties, dysphagia, some nausea and vomiting, and gradual and progressive parenchymal failure of the heart or kidneys. They first ran onto the two sisters, and in a retrospective history were able to obtain from the Wood Wisconsin Veterans Administration Hospital the autopsy material of the father, who had died in 1938 of proved amyloid disease.

At present, at the University of Michigan, the dermatology and neurology sections of medicine are studying a group of Mennonites of Swiss extraction who live in Indiana. They have worked up reports on approximately 300 people in the colony. Their interest was aroused when a patient presented herself to the university and was found to have floating opacities in the eye. The opthalmologist had seen some patients with amyloid disease who had floating opacities, keratitis, and so on. A gingival biopsy was done, and the diagnosis of amyloid disease was made. The patient said that her sister also had some difficulty with vision; when she was studied she was also proved to have amyloid disease.

Electrophoretic patterns were taken on these two patients and the investigators noted a "blip" between the alpha-two and the beta globulin. They considered this to be alpha-three globulin. The pattern was consistent, and through the help of the Mennonite church, they went into Indiana and drew electrophoretic patterns on approximately 300 people. This included adolescents and infants. They found this same electrophoretic pattern in approximately 80 per cent of some family lines, seemingly on a simple Mendelian dominant type of progression. They are postulating that amyloidosis is an inherent disease of mesenchymal tissue, and that eventually the people who have this inherent defect in their tissue will develop primary amyloidosis. They have taken a number of patients from this colony to the University of Michigan and have performed biopsies, and have proved primary amyloidosis, asymptomatic at the time.

Dr. Delp: In July of this year we had admitted here another patient who presented herself with evidence primarily of disease of the cardiovascular system (congestive failure and a gallop rhythm) who had no logical explanation for the etiology of this congestive failure. She had diarrhea, and again I will have to admit that she had had electroshock therapy two years before.

Perhaps because this fitted into the same pattern that we have all stumbled on before, we did make the diagnosis. This patient had involvement of all of the salivary glands, and a biopsy of those glands revealed the diagnosis quickly.

So this is not exactly a rarity, and because of the rather interesting phenomenon that Dr. Christianson has just discussed, I was determined that we would

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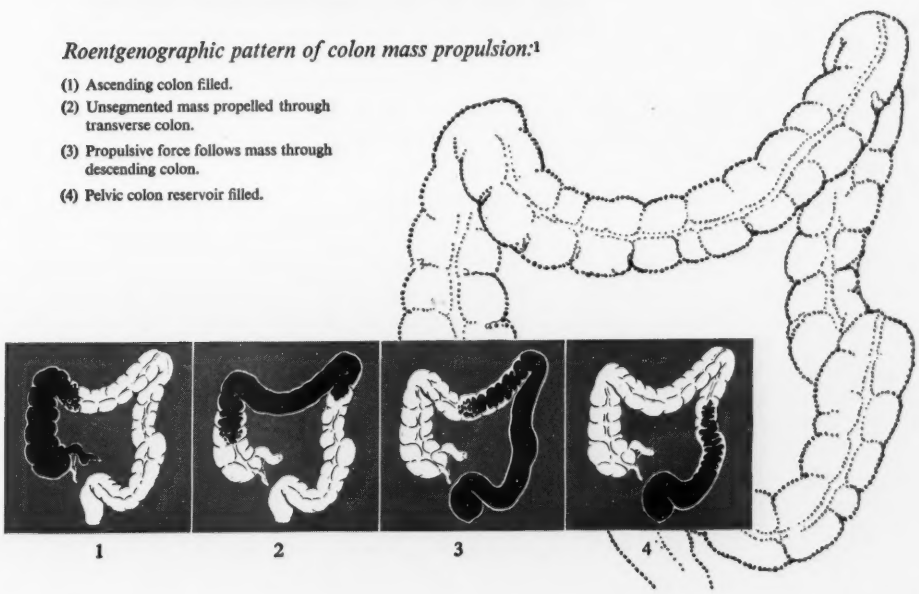

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SEARLE

present this case. Such patients usually present themselves with signs of renal disease, such as nephritis or the nephrotic syndrome, but manifesting either an enlarged liver or cardiovascular findings for which one can find no decent reason. Now I take into consideration the fact that congestive failure does occur in some patients with acute glomerulonephritis, and the subacute forms of the nephrotic syndrome, but this is not very common. So this, as Mr. Chaney indicated, should be a clue. I think the gastrointestinal symptoms which in some respects appear to be similar to the sprue-like syndrome we see in other infiltrative lesions of the small intestines are probably also equally important if we are going to recognize this entity.

PATHOLOGICAL ANATOMICAL DIAGNOSIS

Primary systemic amyloidosis involving arteries and arterioles throughout the body with interstitial amyloid deposits in the heart, spleen, liver, stomach and duodenum, appendix, adrenals, kidneys, ureters, uterus, and ovaries (history of progressive edema beginning six months before death with anasarca and multiple petechiae for three months before death).

Healed infarct of the spleen.

Anasarca with 4500 ml. in the peritoneal cavity, 1200 ml. in each pleural cavity, and 250 ml. in the pericardium.

Multiple petechial hemorrhages in skin, buccal mucosa, left conjunctiva, stomach, ileum, colon, renal pelvic mucosa, and urinary bladder.

Hyperplasia of right femoral bone marrow, slight.

Acute passive congestion of lungs and liver.

Acute hemorrhagic colitis involving the ascending and transverse colon (history of uremia for 12 days before death).

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A booklet on rheumatic fever in children, prepared to supply information to parents, was published recently by the Children's Bureau, U. S. Department of Health, Education, and Welfare. The bulletin, entitled "The Child with Rheumatic Fever," may be secured from the Government Printing Office, Washington 25, D. C.

DEATH NOTICES

JOHN T. KENNEDY, M.D.

Dr. J. T. Kennedy, 77, Blue Mound physician for 51 years, died at Fort Scott on December 16 after a sudden illness. He was graduated from University Medical College, Kansas City, in 1904 and immediately began practice in Blue Mound. He was an active member of the Linn County Medical Society, had served several terms as coroner, and was a member of the Blue Mound city council for 45 years.

EDWIN MCCORMICK IRELAND, M.D.

Dr. E. M. Ireland, 74, a retired physician at Pratt, died at a Topeka hospital on January 10. Dr. Ireland was graduated from the University Medical College, Kansas City, in 1910 and received his Kansas license the same year. He practiced in Coldwater and Protection before entering the service during World War I, then opened an office in Coats, where he continued to practice until his retirement.

EDWARD CHESTER PETTERSON, M.D.

Dr. E. C. Petterson, 61, a member of the Central Kansas Medical Society, died at the Plainville Hospital, of which he was chief of staff, on January 14. He had practiced in Plainville since 1936. Dr. Petterson was graduated from the University of Kansas School of Medicine in 1920 and started practice at Palco. He had served as health officer and coroner of Rooks County.

WILLIAM LEWIS BORST, M.D.

Dr. W. L. Borst, 83, Topeka physician for 35 years, died on January 21. He was a member of the Shawnee County Medical Society. Dr. Borst was graduated from Kansas Medical College, Topeka, in 1900 and practiced for periods of time in Republic, Meriden, McLough, and Belpre before moving to Topeka.

United States hospitals cared for 20,345,431 patients in 1954, more than in any previous year, the American Hospital Association announces. This was an increase of 161,604 over 1953's total.

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1. Rosenberg, S. and Oster, K. A., "Gelatine in the Treatment of Brittle Nails," *Conn. State Med. J.* 19:171-179, March 1955.
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PHYSICIANS' ACTIVITIES

Dr. Howard E. Snyder, Winfield, will speak on "Civilian Aspects of Mass Casualties" at a sectional meeting of the American College of Surgeons to be held in Colorado Springs, March 5-7.

Dr. Dale C. Smith has announced the opening of an office in Mission. A graduate of the University of Kansas School of Medicine in 1945, Dr. Smith served two years in the Army in World War II, practiced in Fredonia for three years, and recently completed a three-year residency in ophthalmology at the Mayo Clinic.

Dr. A. M. Tocker, Wichita, has been elected an associate member of the American Association for Thoracic Surgeons.

Dr. Walter J. Pettijohn, Russell, was recently named by Governor Fred Hall as a member of the Kansas Commission on Alcoholism, succeeding **Dr. Karl Voldeng,** Wellington, who had asked that he not be considered for reappointment.

Dr. Andrew M. Shelton, formerly of Mooreland, Oklahoma, has opened an office in Jetmore. Dr. Shelton, a graduate of Johns Hopkins Medical School in 1951, had a two-year residency in surgery at University Hospital, Little Rock, and practiced in Rockdale, Texas, before going to Oklahoma.

Dr. James G. Gaume, an Ellinwood physician, went to San Antonio last month to serve as research associate physiologist in the department of space medicine, a civilian adjunct to the Air Force. He had served as a flight surgeon from 1942 to 1946 and was introduced to the field of space medicine at that time.

Dr. Jerome S. Menaker, Wichita, presented a paper, "Carcinoma of the Uterus," at a recent meeting of the Southern Medical Association in Houston.

The secretary of the Kansas State Board of Health, **Dr. Thomas R. Hood,** was named to a three-year term on the executive board of the American Public Health Association at the organization's meeting in Kansas City recently. He is also chairman of the Maternal and Child Health Committee, State and Territorial Health Officers Association.

"Man of the Year" was the title conferred on **Dr. Michael W. Scimeca,** Caney, last month. The title was conferred by the student body of Caney High School.

Dr. Frank A. Dlabal, Wilson, is now maintaining an office in Sylvan Grove also and will see patients there two days a week.

After six months of practice in Russell, **Dr. John W. Weigel** reported last month for duty with the Army at Brooke Army Medical Center, Fort Sam Houston, San Antonio. He plans to return to Russell on completion of the Army duty.

The work a community can do in helping solve heart and cancer problems was outlined by **Dr. Haddon Peck,** St. Francis, at a meeting of the Thomas County Health Council in Colby on January 10.

Dr. Ralph R. Reed, who formerly practiced in Beloit in association with **Dr. H. B. Vallette,** has opened an office in Lawrence.

Dr. Robert Hughes, Russell, announces that **Dr. Wendale E. McAllaster,** formerly of Kansas City, Missouri, is now associated with him in practice. Dr. McAllaster was graduated from the University of Kansas School of Medicine in 1954 and served his internship at St. Mary's Hospital in Kansas City.

Dr. Arnold Greenhouse, who recently completed a three-year residency in internal medicine at the University of Kansas Medical Center, is now practicing in Garden City in association with **Dr. H. M. Wiley.**

Dr. Harry R. Custer, Colby, recently completed the examination of the American Board of Surgery and is now a diplomate of that board.

A dinner party honoring **Dr. James A. Simpson,** Salina, was given at the Salina Country Club on January 19 by the Saline County Medical Society. Dr. Simpson recently completed 50 years of practice.

Dr. Alex Scott, formerly of Belleville, recently began practice in Junction City in association with **Dr. C. V. Minnick.**

Dr. John I. Waller, Halstead, recently became a fellow of the American College of Surgeons.

Dr. R. Dale Dickson, Topeka, has been invited

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to present a paper on histamines at the Southwest Allergy Forum at Beaumont, Texas, early in April.

Dr. Conrad M. Barnes, Seneca, is serving as a member of the executive committee of the 1956 Crusade for Freedom.

Dr. E. Ernest Johnson, Norton, has become a member of the surgical staff at the state sanatorium at Norton.

Announcement is made of the marriage of Mrs. Katherine Park Gilliland, Topeka, and **Dr. Lucien R. Pyle,** Topeka, a past president of the Kansas Medical Society. The ceremony was performed at the First Presbyterian Church in Topeka on February 3.

A certificate of merit from the Sedgwick County Cancer Society was presented last month to **Dr. J. P. Berger,** Wichita, for outstanding volunteer service in the fight against cancer.

Dr. Ralph R. Reed, who has practiced in Beloit for the last year and a half, has moved to Lawrence and is practicing there in association with **Dr. L. K. Zimmer.**

Obstetrical Society Meeting

A meeting of the Kansas State Obstetrical Society will be held at the office of the Sedgwick County Medical Society, Wichita, on Thursday, March 1. The meeting will begin in mid-afternoon and will be concluded with a dinner. Dr. E. Stewart Taylor, professor of obstetrics and gynecology, University of Colorado Medical School, will be guest speaker.

Another meeting of the group is scheduled for Tuesday, May 1, at Topeka, a dinner session with an evening program. Guest speaker on that occasion will be Dr. William C. Keettel, of the University of Iowa Medical School.

All members of the Kansas Medical Society are eligible to become members of the Kansas State Obstetrical Society. Checks for dues, $5.00, may be sent to Dr. Edward Crowley, secretary-treasurer, 435 North Hillside, Wichita.

Two-thirds of all maternal deaths can still be considered preventable, according to the Maternal Mortality Committee of the American Academy of Obstetrics and Gynecology on the completion of a study of the matter. It was the opinion of the committee that 1,723 of the 2,610 maternal deaths in the United States in 1952 would not have occurred if ideal care had been available for all expectant mothers.

Nominations for 1956-1957

A meeting of the Nominating Committee of the Kansas Medical Society was held in Salina in December, and the following names were proposed for the various offices to be filled by vote of the House of Delegates at the annual session in May:

FOR PRESIDENT-ELECT

Dr. Barrett A. Nelson, Manhattan

FOR FIRST VICE-PRESIDENT

Dr. Cyril V. Black, Pratt
Dr. Thomas P. Butcher, Emporia
Dr. Grant R. Hastings, Garden City
Dr. Dwight Lawson, Topeka
Dr. Glenn R. Peters, Kansas City
Dr. Robert Sohlberg, Jr., McPherson

FOR SECOND VICE-PRESIDENT

Dr. Cyril V. Black, Pratt
Dr. Thomas P. Butcher, Emporia
Dr. Grant R. Hastings, Garden City
Dr. Dwight Lawson, Topeka
Dr. Glenn R. Peters, Kansas City
Dr. Robert Sohlberg, Jr., McPherson

FOR CONSTITUTIONAL SECRETARY

Dr. George E. Burket, Jr., Kingman

FOR TREASURER

Dr. John L. Lattimore, Topeka

FOR A.M.A. DELEGATE, 1957-1958

Dr. Lucien R. Pyle, Topeka

FOR A.M.A. ALTERNATE, 1957-1958

Dr. Norton L. Francis, Wichita
Dr. H. P. Jones, Lawrence
Dr. Edward J. Ryan, Emporia

The list is presented now in accordance with provisions of the Constitution and By-Laws. Additional nominations, of course, may be made from the floor at the time of the election.

Newspaper Published by Upjohn

A new publication, a weekly newspaper for physicians and their associates in the medical profession, was introduced on January 2 by the Upjohn Company, Kalamazoo. The paper is called *SCOPE Weekly* and is prepared by Physicians News Service, Inc. It contains reports of events and developments in medicine and science from regional, national, and international sources.

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THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

Bills that have been hanging fire in Senate and House committees for over a year finally are getting attention as the administration pushes its program for broader and more uniform medical care for the families of servicemen.

A new version of a bill was dropped in the hopper on the opening day of this session by Chairman Carl Vinson of the House Armed Services Committee. It was designed in part to supply answers to a number of questions growing out of earlier versions sponsored by the Defense Department. Actually it raised more questions, which only hearings and testimony from expert witnesses and debate on the floor of Congress can answer.

The bill (H. R. 7994) authorizes, as a matter of right, broad medical care for dependents of the armed forces as well as of Coast Guard, Public Health Service, and Coast and Geodetic Survey personnel serving on active duty. (The bill would authorize health insurance only for dependents of latter three services.) Separate bills have been introduced in the past providing medical care for dependents of Coast Guard, PHS and Geodetic Survey, but this marks the first time they are brought into the same bill with military personnel.

In provision of services, the bill has no surprises over its predecessors. It calls for diagnosis, treatment of acute medical and surgical conditions, treatment of contagious diseases, and maternity and infant care.

On another point of major interest to physicians, the bill drops out all mention of the home-town medical care plan, which was a part of Mr. Vinson's earlier bill. That bill contemplated use of civilian hospitals and doctors for those dependents who were not near military medical facilities and who had not taken out health insurance, with the government paying part of the cost.

Another area of almost certain debate in the latest bill is the insurance features. There are these main points:

1. A serviceman may elect to rely entirely on the chance of finding space available in a military hospital or clinic for his family, or he may choose protection through an insurance plan.

2. The family deciding on insurance has its choice of going to a military hospital or using civilian resources. The uninsured family could be charged by the military for out-patient care, and would have to pay subsistence costs while in the hospital.

3. A serviceman taking insurance would pay 30 per cent of monthly premiums for a basic plan covering his wife and children, and the entire premiums for coverage of dependent parents and parents-in-law. Parents and parents-in-law who found space in a military hospital, however, would be admitted on the same basis as wives and children.

4. Catastrophic-type coverage, at additional premium.

5. To take care of long term illnesses, the bill provides for transfer of dependents to military facilities once they have used up benefits in an insurance plan. Or if such transfer isn't feasible, the government could pay the additional costs for private care.

The bill was introduced before the Defense Department had completed a survey of Blue Shield, Blue Cross, and commercial plans to determine to what extent they could provide care under the bill. Conceivably the survey could further change the shape of an already much-revised piece of legislation.

President Eisenhower in his State of the Union message summed up the case for dependent medical care this way: "Much has been done to attract and hold capable military personnel, but more needs to be done." He also broadly outlined administration plans in the health field, with emphasis on more money for research and federal aid to medical schools and to private research facilities for construction. With bipartisan bills along this line already before Congress, these proposals may move right along before adjournment in mid-summer.

However, Congress might decide that for this year medical schools should settle for the $90 million of Ford Foundation money being made available to private schools to help strengthen teaching staffs.

By the same token, there was some question just how much Congress would vote for Hill-Burton hospital programs this session in the light of the $200 million Ford grants to some 3,500 non-profit hospitals.

A recent Public Health Service report indicates that states are now showing less preference for "public" Salk vaccine programs than they did a few months ago. The sixth allotment marked the high point in "public" preference. Then came a slight but steady decline.

Annual meeting, Kansas Medical Society, Topeka, April 29-May 3, 1956.

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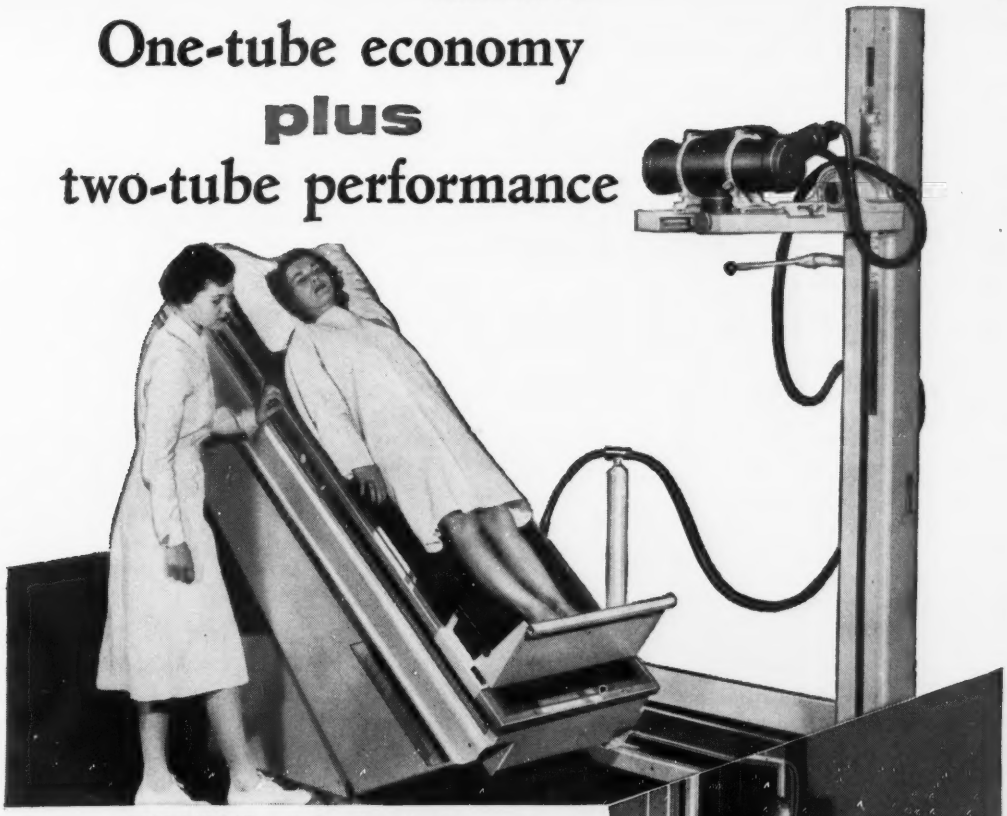

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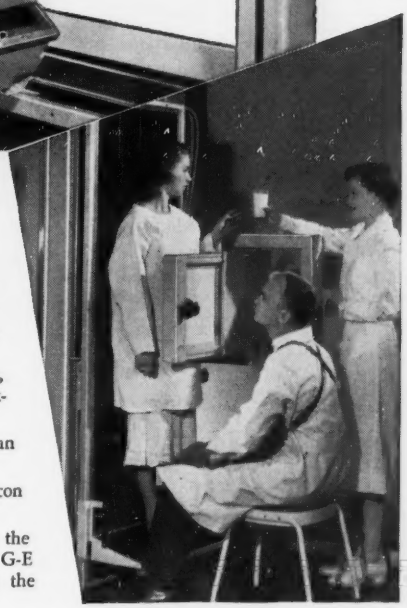

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COUNTY SOCIETIES

The Shawnee County Medical Society, at a meeting held in Topeka on January 3, voted an assessment for each member in 1956 as a means of securing a sizable contribution for the American Medical Education Foundation. Active members will pay $15 each, associate members $7.50. Emeritus members are exempt from paying the assessment.

The scientific presentation of the evening was given by Dr. Robert J. Crossen, St. Louis, who spoke on "Vaginal Plastic Operative Work."

Dr. Robert Cotton, Topeka, was elected to membership.

Dr. Leland Speer, Kansas City, was elected president of the Wyandotte County Society at a meeting held in November and took office on January 7 at a dinner meeting at the Town House Hotel. Others elected are: vice-president, Dr. William P. Williamson; secretary, Dr. Francis J. Nash, and treasurer, Dr. James B. Pretz.

A seminar on legislative and medicolegal subjects was a feature of the January 3 meeting of the Sedgwick County Society in Wichita. Mr. Harvey T. Sethman, Denver, executive secretary of the Colorado State Medical Society, spoke on "Your Legislative Problems," and Mr. William Tinker, Wichita attorney, discussed "Recent Trends in the Law as it Relates to the Practice of Medicine."

The following physicians, elected at a previous meeting, took office at that time: president, Dr. J. P. Berger; vice-president, Dr. William J. Reals; secretary, Dr. J. H. Holt; treasurer, Dr. Dean A. Huebert. Serving on the Board of Directors are: Doctors W. P. Callahan, Jr., H. C. Clark, G. F. Gsell, L. K. Crumpacker, H. F. O'Donnell, C. L. Woodhouse, G. E. Cowles, G. L. Thorpe, and L. E. Vin Zant.

Dr. L. J. Beyer, Lyons, was elected president of the Rice County Society at a meeting held recently in Lyons. Dr. James T. Grimes, Lyons, was named vice-president, and Dr. Preston E. Beauchamp, Sterling, was chosen secretary-treasurer.

Members of the Saline County Society were hosts to the Golden Belt Medical Society at a meeting held at the Lamer Hotel, Salina, on January 12. Dr. Walter Mau, Topeka, spoke on "Emergency Urology Problems," after which a group of pediatricians cooperated to discuss common illnesses and problems of infants and children. Dr. Lucius E. Eckles, Topeka, was moderator. Participants were Dr. Donald Diefendorf, Waterville; Dr. O. L. Martin, Salina; Dr. Harry O'Donnell, Junction City, and Dr. Forrest Taylor, Clay Center.

Dr. Darwin L. Richardson, Minneola, entertained members of the Comanche-Kiowa-Clark Society at a dinner meeting at Minneola recently.

Members of the Marion County Society entertained their wives at a dinner meeting at Marion on December 19. An octet from Sacred Heart School, Wichita, presented a musical program.

A meeting of the Barton County Society was held at Great Bend on January 9. Mr. Rueben M. Dalbec, executive assistant of the Kansas Medical Society, and Mr. Hart Workman, attorney for the Kansas State Board of Social Welfare, spoke on different phases of medical care for the indigent. A question and answer period followed.

Officers of the Crawford County Society for 1956 were elected at a meeting held at Pittsburg on December 29. The following were named: president, Dr. William T. Braun, Pittsburg; vice-president, Dr. Jack D. Walker, Girard; secretary-treasurer, Dr. Clifford B. Newman, Pittsburg.

Dr. David S. Ruhe, head of the Department of Audio-Visual Education at the University of Kansas Medical Center, was speaker at the January 17 meeting of the Wyandotte County Society at Battenfeld Auditorium. He discussed "Color Television and the Medical Profession."

A meeting of the Wilson County Society was held at the Methodist Church in Neodesha on January 11 with members of the Auxiliary as dinner guests. Mrs. Goldena Olinger, county director of welfare, was speaker of the evening.

A meeting of the Montgomery County Society was held in Coffeyville on January 18. Dr. J. E. McDonald, Tulsa, a member of the legislation committee of the American Medical Association, spoke on social security proposals to be considered by Congress.

The A.M.A. clinical session in Boston in December drew a total registration of 8,637, including 3,779 physicians. Corresponding figures for the Miami meeting in 1954 were 7,707 and 3,253.

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Psychomotor Epilepsy

ROBERT C. BRANDMEYER, M.D., ***Panama Canal Zone***

Psychomotor seizures may be defined as episodes of complex, usually well coordinated, automatic motor activity produced as the result of an epileptic discharge within the central nervous system. The term "automatic activity" is used here in the sense that it is motor behavior over which the individual has no conscious control. The question of whether the patient is actually conscious or unconscious during the seizure awaits the determination of more generally agreed upon qualifications for these terms. However, it is known that the state of consciousness is altered at least to the extent that the patient has lost his capacity for making a durable memory record for events occurring during the course of an attack, and he has total amnesia for all that has transpired.[28]

There is no general pattern of behavior characteristic of all cases of psychomotor epilepsy, although there is usually some consistency in the manifestations of the individual attacks occurring in a single patient. The periods of automatic behavior and amnesia may be quite short in duration, lasting only about 60 seconds, or they may continue for several hours.[9] Again there is usually some consistency in the time interval involved in individual cases.

During the shorter episodes the patient may exhibit relatively unelaborate patterns of motor activity, a common example being masticatory movements with smacking of the lips and swallowing while at the same time giving a general appearance of confusion. During the prolonged attacks a patient may exhibit extremely complex and bizarre behavior such as running in circles, disrobing, or engaging in some well coordinated activity which appears purposive but irrational or inappropriate for the time and place. The patient may continue with something he was doing at the onset of the seizure but not respond to a command to alter the pattern of his activity. Speech may be slurred but is usually not affected except in content, which is often obscure or meaningless. In some cases the automatic behavior is characterized by an emotional display of rage and violence, during the course of which the patient may be aggressive, may attack other persons, or engage in other forms of destructive activity. Observation of clonic or tonic convulsive movements during or preceding a psychomotor attack has been reported in a few cases, but they are relatively rare.

The only clinically observable accompaniments which consistently occur in all episodes of psychomotor automatism are a period of obvious confusion at the end of the episode and total amnesia for the experience. Frequent though not invariable features are those of wandering about with a general appearance of confusion, together with an apparent inability to relate the environment and events to past experience.

An aura may or may not occur at the onset of the seizure. If present it may be one in which the patient experiences some non-specific sensation such as a feeling of fullness of the head, an abnormal epigastric or intra-abdominal sensation, or simply an awareness of an impending seizure. More often, however, the attacks of psychomotor automatism are ushered in by elaborate and complex psychical phenomena. These may consist of sudden involuntary vivid recollections of past events, perceptual illusions, hallucinations, or illusions of interpretation in which there is a distortion of the patient's ability to relate present to past experience which results in feelings of unusual familiarity, absurdity, or fearsomeness about his surroundings.

In about one-third of all cases of psychomotor epilepsy there are no clinical manifestations of epilepsy other than the psychomotor seizures. The remaining approximate two-thirds of these patients also have grand mal seizures apart from the psychomotor attacks. While petit mal seizures and other epileptic variants may occur clinically in a patient with psychomotor epilepsy, such combinations are rare.[13]

In view of the absence of the more generally recognized signs of grand mal and petit mal epilepsy during nearly all episodes of psychomotor automatism, together with the tendency of the attacks to resemble psychiatric rather than primarily neurological disturbances, it is surprising that recognition of the entity as a type of epilepsy occurred as early as it did. In 1888, Hughlings Jackson suggested that what appeared in some patients to be periodic episodes of psychotic or hysterical behavior might be the manifestations of a particular type of epileptic seizure. Jackson confirmed this suspicion to his own satisfaction, and by a process of clinicopathological correlation concluded that the focus of the discharging

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This is one of 11 theses, written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school. Dr. Brandmeyer is now serving his internship at Gorgas Hospital, Panama Canal Zone.

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lesion in psychomotor epilepsy was located in the temporal lobe.[17]

In the interval between the clinical recognition of psychomotor epilepsy and the development of an adequate method of electroencephalographic study there was no uniformity of opinion as to whether the psychomotor attacks represented a certain type of seizure, manifestations of postseizure phenomena, or substitutes for seizures. Electroencephalographic exploration has resulted in recent years in the determination of definite correlations between fluctuations in electrical potential over areas of the human brain and the clinical picture of psychomotor epilepsy.

In 88 per cent of patients with clinical psychomotor seizures there is a detectable focus of interseizure discharge which is located in the anterior temporal region either unilaterally or bilaterally. A focus of abnormal interseizure activity in this location has been given the name "psychomotor focus." Also, a type of seizure discharge distinct from the rapid frequency of grand mal and the alternate slow and fast frequency of petit mal has been found to be typically associated with psychomotor seizures and has been labeled the "psychomotor seizure pattern" of discharge. It is composed of cortical action potentials which are of high voltage and slow frequency, from three to six per second.[11, 12]

With the use of activation techniques such as hyperventilation or intravenous injection of small doses of metrazol, it has been possible to induce clinical seizures in patients while electroencephalographic recordings are being taken. These records and observations usually reveal a typical sequence of events. The psychomotor interseizure focus is usually composed of random positive spikes. If the spikes remain confined to the temporal lobe, there are no clinical seizure manifestations. If a clinical seizure is impending, the spikes occur with progressively increasing frequency and are finally replaced by the high voltage three to six per second waves which spread to all cortical areas. It is at this point that clinical manifestations usually appear, although in some instances this type of dysrhythmia may continue for several seconds without simultaneous clinical manifestations.[11]

After the period of generalized discharge, the electroencephalographic record typically becomes flattened in all leads and is characterized chiefly by a relative absence of detectable cortical activity, a period of so called postictal exhaustion or paralysis.

There may be no clinically apparent change in the nature of the clinical seizure as the generalized discharge pattern is replaced on the record by the postictal period of cortical inactivity. The automatism gradually merges into a period of confusion and finally to a full recovery of conscious processes as the record shows return to a normal electrical activity.[13]

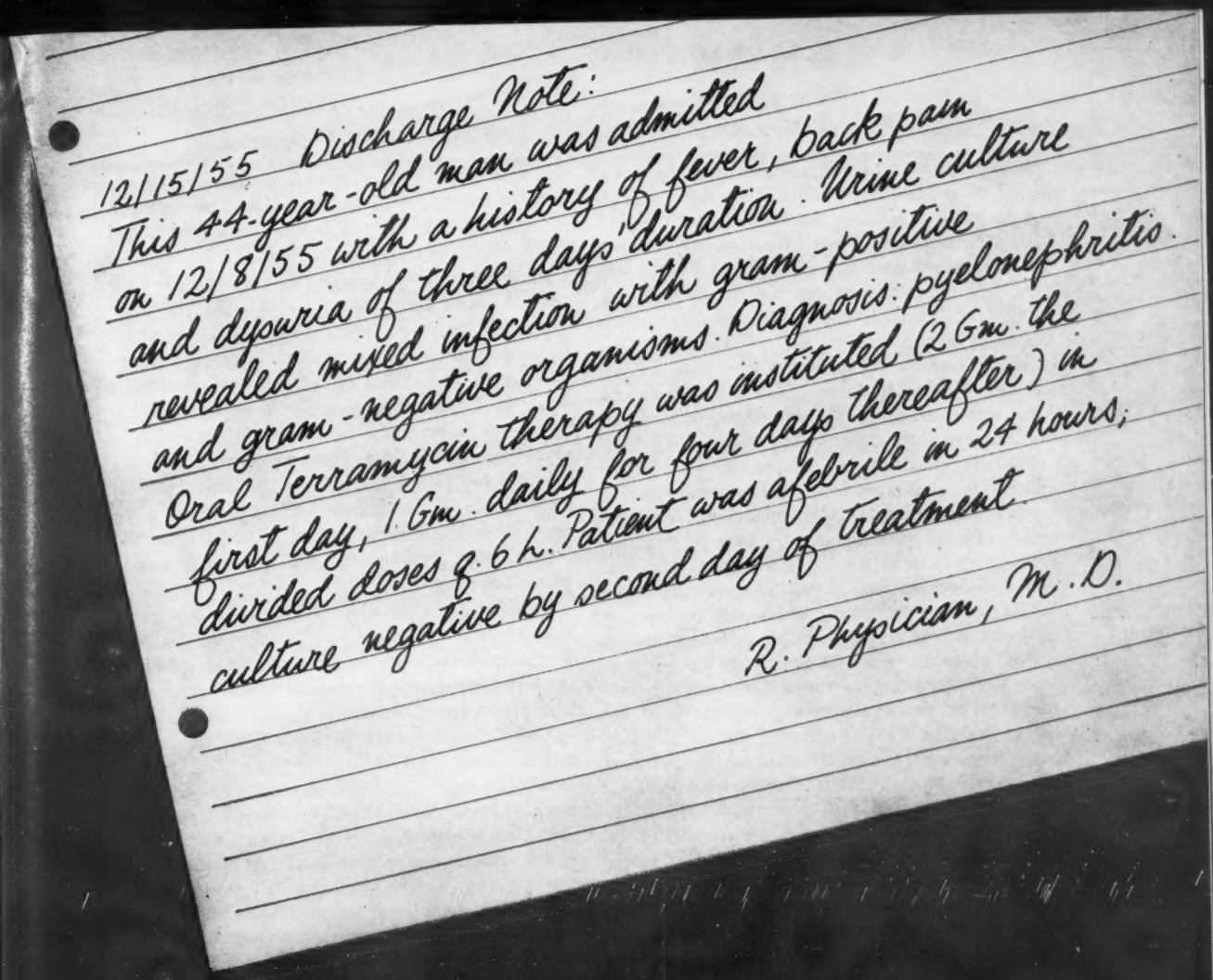
These electroencephalographic findings of a psychomotor focus and a psychomotor seizure pattern, while demonstrable in the majority of cases, are not invariably present. In an occasional case of psychomotor epilepsy the only demonstrable discharging cortical focus is located in the frontal lobe (less than one per cent) or in the occipital lobe (less than one per cent),[13] and recently there have been reported two cases of clinical psychomotor epilepsy in which the pattern of seizure discharge was that of the petit mal type.[8]

Not all cases of epilepsy with a demonstrable temporal lobe focus fall into the clinical category of psychomotor epilepsy. However, in cases with a discharging focus in the anterior one-third of the temporal lobe, 90 per cent have clinical psychomotor automatism. In the remaining 10 per cent there may be only grand mal or other varieties of clinical seizures associated with the demonstrable psychomotor focus.[13, 17]

It has recently been shown that the temporal lobe discharge in psychomotor epilepsy may be primary, with the focus of origin of epileptogenic discharge located in the anterior temporal lobe, or the temporal lobe discharge may be secondary to a primary epileptogenic focus located within the deeper structures of the brain stem.[12, 14, 26] The pattern of ictal discharge is identical in both types.

There is a primary focus of discharge on one temporal cortex in about 75 per cent of the cases. In these there is abnormal interseizure activity over only one temporal cortex, or predominantly on one side with secondary extension of the abnormal discharge to the opposite temporal cortex. In the remaining 25 per cent there is a primary focus of discharge in the higher brain stem. The interseizure discharge in these cases is characterized by a simultaneous synchronous discharge in both temporal lobes, or by a discharge occurring alternately in first one and then the other temporal lobe.[14, 18, 27, 29] A neuron system within the higher brain stem which is functionally related to the anterior temporal cortices has been demonstrated in man,[32] and the production of epileptiform spikes in the anterior temporal lobes bilaterally, secondary to stimulation of the septal nuclei and specific local areas of the thalamus and basal ganglia, has been accomplished in studies with cats and monkeys.[1]

It is believed that in all attacks of psychomotor automatism both the cortical and brain stem structures become involved, the difference being in the order of involvement.[2, 13, 17, 29] The development of the automatism and amnesia apparently occurs subsequent to the involvement of the midbrain structures.[29]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In the case of a primary temporal lobe focus, the period of automatism is initiated by the spread of the discharge from the temporal cortex to the midbrain structures with which it is connected. Prior to this extension, the local abnormal activity in the temporal lobe either may or may not result in the production of an aura. In cases with a primary focus in the brain stem structures and only secondary discharge in the anterior temporal lobes, there is never an aura, the initial discharge of the epileptogenic focus within the deeper structures being immediately productive of the automatic state.

Electroencephalographic studies of psychomotor epilepsy have shown, then, that the production of psychomotor automatism is almost invariably associated with the presence of a primary or secondary discharging focus in the anterior temporal lobe, and with a distinct rhythmic pattern of seizure discharge. While psychical phenomena which often precede psychomotor attacks are apparently produced as the result of abnormal neuronal activity in the temporal lobe, the production of the actual automatic state seems to be dependent upon involvement of the higher brain stem nuclei to which the anterior temporal lobes are functionally attached. Studies have also revealed that clinical automatism may occur not only during the period of generalized seizure discharge but also may extend into the postictal period while there is still an alteration of function of the central nervous system consequent to the discharge.

Further speculation as to the nature of the functional alteration in the central nervous system responsible for the production of psychomotor automatism has come from the experimental studies of Dr. Wilder Penfield and his associates. They observed the effects of direct electrical stimulation of the human cerebral cortex during the course of surgery under local anesthesia.[24, 25, 26, 27, 28, 29]

Electrical stimulation of the temporal lobe cortex may produce any of the complex psychical phenomena which frequently occur as an aura at the onset of a psychomotor seizure. Some patients experience sudden and involuntary recollections of happenings in their recent or distant past. These memories are much more vivid than those the patient could produce voluntarily. Patients describe them as a reliving of the remembered episode in much the same way as a scene is recalled in a dream. There may be auditory or visual hallucinations or both, but the hallucinations are always concerned with a specific past experience. The stimulation results in the production of no new material in the mind of the patient.

Illusions of interpretation may also occur, either as an aura preceding a psychomotor seizure or as the result of electrical stimulation of the temporal cortex. One of the most commonly encountered experiences of this type is the so called déjà vu phenomenon in which patients have a sudden feeling of unreal familiarity with their surroundings and situation, as if they were living through this part of their lives for the second time.

Penfield has suggested that all psychical phenomena produced represent some type of abnormal function of memory mechanisms due to abnormal stimulation either by a stimulating electrode or an epileptic discharge. In some instances abnormal temporal lobe stimulation seemingly produces an activation of specific memory patterns. In other instances it results in an aberration in the processes of interpretation of the environmental situation because of some distortion of the patient's ability to accurately relate present to past experiences.

From these speculations Penfield has suggested that the temporal cortex and the neuron complexes in the midbrain with which it is related constitute the memory organ of the central nervous system, and that psychomotor automatism is produced as the result of ictal and postictal paralysis of the function of memory recording.[27, 29]

While a more complete understanding of the exact mechanism of production of psychomotor automatism is lacking at present, there has been much interest in recent years in the relative incidence of the disease and the development of methods of treatment. Recent estimates of the incidence of psychomotor epilepsy, either alone or in conjunction with other forms of clinical seizures, suggest that they occur in 25[13, 27] to 40 per cent[2, 19] of the epileptic population. These figures are much higher than those of only a few years ago. It has been suggested that the increase is due principally to a more general awareness of the variable manifestations of the entity, together with the development of electroencephalographic techniques to detect an abnormally discharging focus in a higher percentage of cases.

Psychomotor epilepsy occurs most commonly in adults, with a slightly greater incidence in males than in females. The first attack usually occurs after the age of 20 years and rarely earlier than 10 years of age. In over 30 per cent of the cases the first attack occurs sometime during the third decade of life.[13, 33]

Studies of etiological factors responsible for the production of a discharging focus in cases of psychomotor epilepsy have resulted in the classification of 75 per cent of the cases as idiopathic. In those cases in which etiology has been presumed, trauma, encephalitis, birth injury, brain tumors, and brain abscesses have been designated as more frequent causes, while vascular accident, arteriosclerosis, mastoiditis,

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and meningitis have been believed responsible in occasional cases.[13, 31]

Hereditary factors seem to be of about the same importance in psychomotor epilepsy as in other forms of epilepsy, a positive history of some form of epileptic seizure being elicited from close relatives of these patients five times as frequently as in the general population.[20] In many cases both a transmitted predisposition and brain damage seem to be important contributory factors in the origin of the seizures.[20]

There are certain other clinical conditions which are frequently confused with psychomotor epilepsy. Short episodes of psychomotor automatism are frequently misdiagnosed as petit mal seizures. The clinical points of differentiation are that psychomotor epilepsy usually occurs in adults while petit mal seizures occur most commonly in children. Psychomotor attacks usually occur with a frequency of once a month to once or twice a week, and rarely more frequently than once a day,[9, 13] while petit mal seizures often occur with a frequency of several times a day. Even brief psychomotor attacks usually last for 60 seconds or longer, while petit mal seizures are usually of shorter duration than 60 seconds. There is no period of confusion following petit mal seizures, while this is an invariable accompaniment of psychomotor attacks. There is frequently an aura with psychomotor epilepsy while an aura is almost never associated with petit mal. Finally, electroencephalographic differentiation is usually possible.[9, 16, 33]

Differentiation between psychomotor attacks and psychoneurotic behavior is often difficult, particularly since severe personality disturbances are present in about 50 per cent of patients with psychomotor epilepsy, and of this 50 per cent one-half have a history of psychosis, while positive clinical neurological findings are nearly always absent.[9, 10, 13, 33] Many patients, particularly those with severe neurotic tendencies, are reluctant to admit having experienced psychical phenomena through fear of being considered psychotic. When psychomotor epilepsy is suspected, it is frequently necessary to ask the patient directly about the occurrence of bizarre episodes and reassure him that his seizures are actual experiences and not breaks with reality.

The chief points of differentiation between psychomotor seizures and bouts of psychoneurotic behavior are: (1) Psychomotor seizures are episodic with a fairly well defined beginning and ending. (2) Each attack tends to follow a similar pattern. (3) The attacks are usually unrelated to environmental factors. (4) There may be an aura. (5) There is amnesia for the psychomotor seizure.[9, 16, 33] Eliciting a family history of any form of seizures or a personal history of head injury or convulsions secondary to fever or toxic conditions should also suggest the possibility of psychomotor seizures in an individual whose emotional disorder clouds the clinical picture.[20, 31]

The differential diagnosis between psychomotor epilepsy and hysteria can be most difficult. In both the attacks may be periodic. Hysterical episodes often begin with the resemblance of an aura, and there may be amnesia for the experience. An electroencephalographic diagnosis is frequently not easy, for a small number of psychomotor epileptics retain normal records in spite of activation techniques, while some hysterical individuals show electroencephalographic anomalies similar to those of epileptics. One point of differentiation is that under the influence of hypnosis the hysterical individual may be able to recall his experience during an attack while the psychomotor epileptic cannot. In some cases, only response to therapy will be of assistance in making a final diagnosis.

In the absence of a disease entity making surgical intervention mandatory, such as an operable brain tumor or a subdural hematoma, all authors agree that a medical therapeutic trial should be attempted with cases of psychomotor epilepsy, and elective surgery may be considered only when all else fails.[7, 12, 15, 17, 26] General supportive measures in conjunction with medical therapy should include avoidance of alcohol, physical fatigue, and situations which put an undue emotional strain on the patient, since all of these have been shown to predispose toward attacks.[2, 10] In some patients with severe emotional disorders, a psychiatrically oriented therapeutic and supportive program seems to be the most valuable measure in control of the seizures.

The response of psychomotor epileptics to drug therapy is variable, and treatment with standard preparations must be individualized. In general it may be said that results of medical treatment have been less gratifying in the case of psychomotor epilepsy than with other types of epileptic seizures. Bromides are beneficial in rare instances but have been noted to accentuate personality disturbances in others.[20] Phenobarbital, either alone or in conjunction with other drugs, often results in an increase in frequency of the seizures,[12] although in one series of 25 patients, 17 reportedly showed improvement while taking Dilantin and phenobarbital.[7] Dilantin alone is beneficial in some cases and apparently ineffective in others.[7, 30]

Trials with Tridione, Paradione, and Mesantoin, either alone or in conjunction with other drugs, indicate that they are rarely effective and may cause an increase in the frequency and severity of attacks.[7, 30, 33] Milontin (N-methyl alpha-phenylsuccinimide) has been reported as being of marked value in reducing

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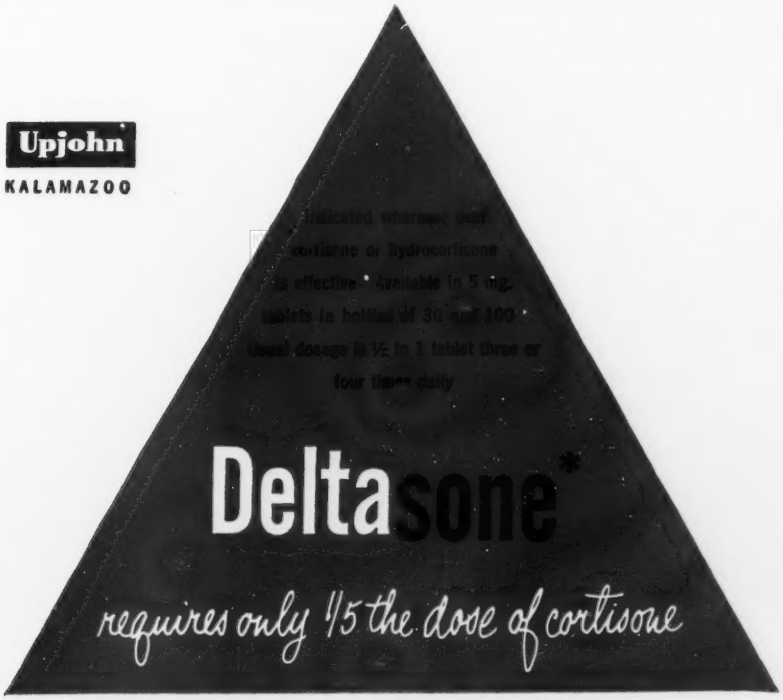

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On 6/3/55 patient was readmitted because of purulent discharge from wound. On 6/3 a hemolytic Staph. aureus (coag. +) was isolated from abscess with the following disk sensitivities: penicillin, 1.5 units; erythromycin, 10 mcg; tetracycline, 10 mcg. Patient was placed on penicillin, 600,000 units b.i.d. for 10 days. On this schedule patient improved but progress was unsatisfactory and wound continued to discharge small amount of purulent material.

On 6/13 penicillin was discontinued and erythromycin started in dosage of 200 mgm. q.i.d. By 6/17 the discharge had stopped and wound was completely healed by 6/19. Erythromycin was continued until the patient was discharged from hospital on 6/21. Temp. was normal throughout hospital stay.

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the incidence of psychomotor seizures,[4] but this drug is also said to have extremely toxic side effects.[30]

Phenurone has been the most effective drug produced with respect to control of psychomotor seizures, but it is also capable of producing a number of alarming side effects which have caused several authors to be reluctant to endorse its use even under close supervision.[7, 12, 30, 33] Among the side effects noted have been severe depressive states, psychosis, extreme agitation and restlessness, leukopenia, aplastic anemia, and severe hepatic damage.

Most authors agree that an initial trial with Dilantin is a preferred procedure, and a combination of Dilantin and phenobarbital may produce good results. A mixture of Dilantin, phenobarbital, and Phenurone has been suggested as effective in some otherwise refractory cases.[12]

When Phenurone is administered, close sypervision is indicated, and the appearance of any of the toxic symptoms listed should indicate discontinuation of the drug.

It has been noticed that effective medical control, regardless of the drug used, produces an exaggeration of psychoneurotic symptoms in some patients.[12]

The presence of multiple types of seizures is apparently a favorable prognostic sign with regard to medical therapy.[7, 12] In cases of the mixed occurrence of grand mal and psychomotor seizures, good results are obtained in the majority of cases with the use of Dilantin alone. The frequency of occurrence of seizures and the age of onset of attacks are not significantly related to the results of drug therapy, but the duration of the seizures prior to institution of therapy seems to be important. If the patient has had seizures for 10 years or more, there is a sharp drop in expected percentage of beneficial results.[7]

Patients with incapacitating and medically refractory cases of psychomotor epilepsy may, in carefully selected cases, be helped by surgical treatment. The surgical procedure followed is the removal of the anterior portion of the involved temporal lobe. The most necessary requirement for elective surgery is, therefore, the electroencephalographic demonstration of a definite cortical focus of interseizure discharge which occurs persistently on only one side, or predominantly on one side with only secondary involvement of the opposite temporal lobe.[2, 15, 17, 26] Recognition of the fact that the temporal lobe discharge in psychomotor epilepsy may be secondary to an epileptogenic focus within deeper structures has explained many earlier surgical failures.[15] The presence of an aura is additional evidence suggesting a primary temporal lobe focus of discharge. The electroencephalographic demonstration of bilateral synchronous temporal lobe discharge at the initial stage of development of a seizure, together with the absence of a history of aura manifestations at the onset of a seizure, constitutes a strong contraindication to temporal lobectomy for alleviation of psychomotor symptoms, even in the presence of known organic pathology in one temporal lobe.[17, 25]

Surgical results indicate also that when features of rage or an intense emotional display are prominent in the seizures, surgery is rarely beneficial.

For most patients who had a definite operable focus in only one temporal lobe and whose seizures had no prominent emotional features, postoperative results have been good. Complete or nearly complete postoperative control of seizures with medical therapy has been reported in 75 per cent of these patients, with some decrease in frequency and severity of attacks in nearly all. The best surgical results were obtained in cases in which electroencephalographic recordings were made during surgery to insure complete excision of the focus of abnormal discharge.

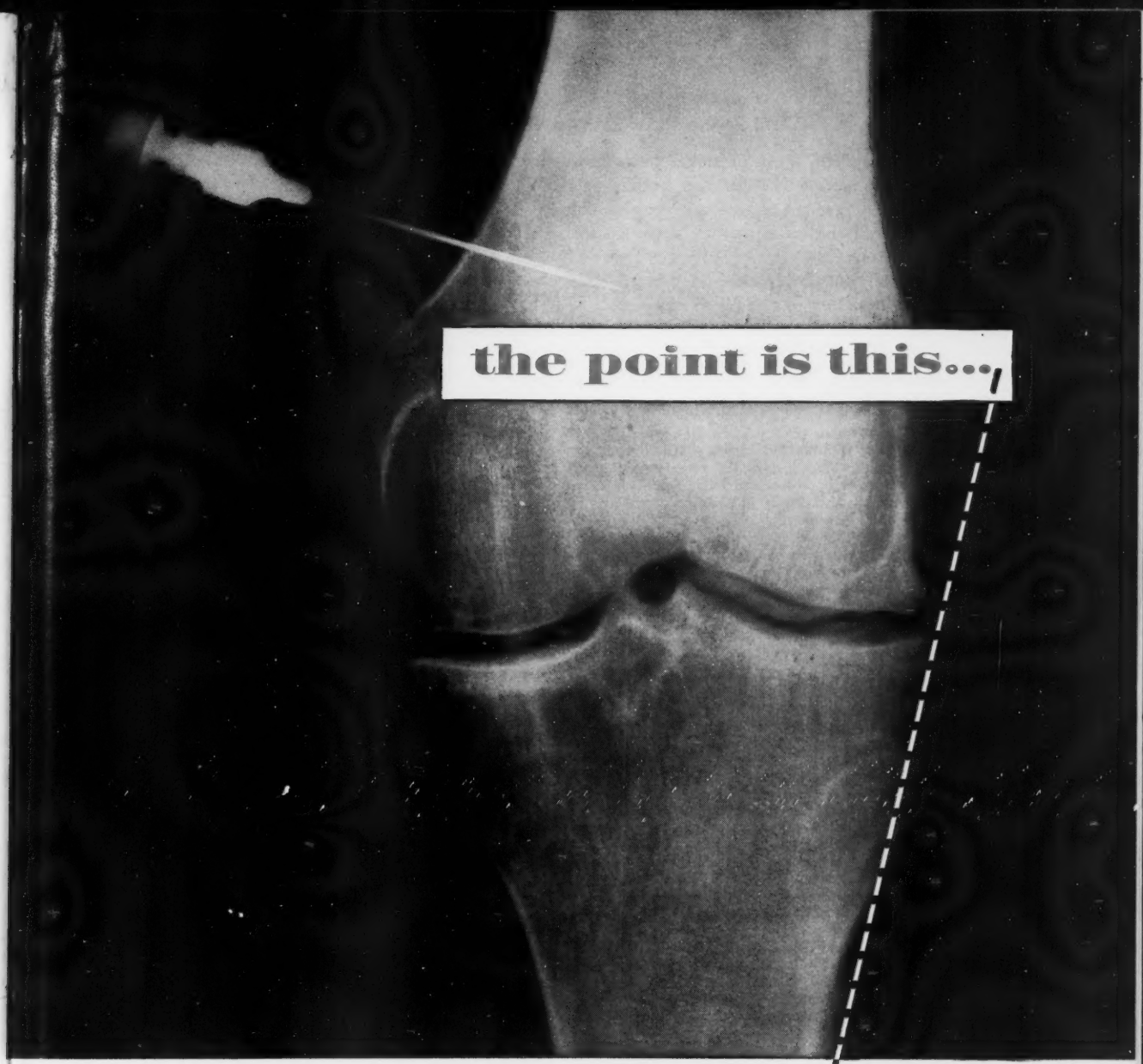
As with medical control, surgical alleviation of seizure symptoms occasionally results in an increase in severity of psychoneurotic tendencies.

Except in cases where the temporal gyration of the optic nerve was severed, producing homonymous hemianopsia, or the receptive speech center was removed, resulting in receptive aphasia, there have been no apparent neurological or intellectual deficits produced by the removal of one anterior temporal lobe.

In only one-third to one-half of the cases was there an anatomical lesion demonstrable either grossly or microscopically at the site of primary epileptogenic discharge in the temporal lobe.

Psychomotor epilepsy has been described as an epileptic variant in which the manifestations of epileptic discharge are those of complex motor activity during a period for which the patient has complete amnesia. The seizures may or may not be associated with psychical phenomena. Psychomotor discharge is typically associated with interseizure random spikes in one or both temporal lobes. The primary origin of abnormal discharge may be either in a temporal lobe focus or an epileptogenic focus within the higher midbrain. There is a typical pattern of seizure discharge associated with the attacks.

Psychomotor epilepsy is frequently mistaken for a psychiatric disorder because of the bizarre nature of symptoms and the high incidence of personality disorder in such patients. A recent increase in interest in psychomotor epilepsy has resulted in the determination of a much higher incidence than was formerly suspected.



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Psychomotor epilepsy seems in general to be more resistant to drug therapy than are other types of epileptic seizures. A program of general supportive and medical therapy is beneficial to some extent in the majority of cases.

Surgical treatment is advocated in selected cases among those refractory to medical management.

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Study of Uterine Cancer

Study of a new method for detecting uterine cancer is now being conducted in eight United States communities by the National Cancer Institute of the Department of Health, Education, and Welfare. The technique, involving examination of cells which have been shed by the uterus, received its preliminary evaluation on a mass basis in studies conducted in Memphis during the past two years.

The department reports that the test is a relatively simple procedure. A specimen of fluid from the vaginal vault is examined for the presence or absence of cancer cells. If cancer cells are present, further study by biopsy establishes a firm diagnosis.

In the Memphis study the test produced a case-finding rate 40 times that observed in the community prior to establishment of the project. In the first 70,000 women tested, 88.3 per cent of the early curable cases discovered were in women in whom the disease had not been previously diagnosed or suspected.

Under revised promotion policies, physicians will be eligible for temporary promotion to the grade of major, lieutenant colonel, and colonel one year sooner than other Army officers, it has been announced by Major General Silas B. Hays, surgeon general. It is estimated that 390 medical officers will be promoted to higher grades before June 30 as a result of the policy.

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ANNOUNCEMENTS

Annual meeting, Atlanta Graduate Medical Assembly, Biltmore Hotel, Atlanta, Georgia, February 20-22.

Harvard University School of Public Health scholarships available for academic year 1956-1957. Closing date for applications, March 1. Address inquiries to 55 Shattuck Street, Boston 15.

Sectional meeting, American College of Surgeons, Colorado Springs, March 5-7. Symposia, panel discussions, cine clinics. Address inquiries to Dr. H. Prather Saunders, A.C.S., 40 East Erie Street, Chicago 11.

Tenth annual symposium on fundamental cancer research, University of Texas M.D. Anderson Hospital and Tumor Institute, Houston, March 29-31. General topic, "Cellular Metabolism of Tumors." Reports on research projects being conducted.

Graduate instructional course and 12th annual meeting, American College of Allergists, Hotel New Yorker, New York City, April 15-20. Address inquiries to Dr. F. W. Wittich, 401 LaSalle Building, Minneapolis 2, Minnesota.

Centennial celebration and scientific congress, Hawaii Medical Association, Honolulu, April 22-29. Scientific program, pageant, traditional Hawaiian feast, planned tours. Address inquiries to 510 South Beretania Street, Honolulu 13, Hawaii.

Teaching seminar, International Academy of Proctology, Drake Hotel, Chicago, April 23-26. Program on anorectal and colon surgery. Physicians invited. No fee. Address inquiries to 147 Sanford Avenue, Flushing, New York.

Annual meeting, American Goiter Association, Drake Hotel, Chicago, May 3-5. Physiology and diseases of thyroid gland. Address inquiries to Dr. John C. McClintock, 149½ Washington Avenue, Albany, New York.

Prize for essay on any subject relating to physical medicine and rehabilitation offered by American Congress of Physical Medicine and Rehabilitation. Contest closes June 1. Address inquiries to 30 North Michigan Avenue, Chicago 2.

First International Congress of Human Genetics, Copenhagen, Denmark, August 1-6. Address inquiries to University Institute for Human Genetics, 14, Tagensvej, Copenhagen, N., Denmark.

Ninth annual meeting, American Association of Blood Banks, Somerset Hotel, Boston, September 3-5. Address inquiries to Miss Marjorie Saunders, 725 Doctors Building, 3707 Gaston Avenue, Dallas, Texas.

Tenth congress, International College of Surgeons, held in conjunction with 21st congress, United States and Canadian sections, Chicago, September 9-13. Address inquiries to 1516 North Lake Shore Drive, Chicago 10.

Scientific and clinical session, American Congress of Medicine and Rehabilitation, Ambassador Hotel, Atlantic City, September 9-14. Open to members of A.M.A. Address inquiries to 30 North Michigan Avenue, Chicago 2.

A meeting of members of the American College of Physicians of this region, originally scheduled for March 23, will be held on March 9 in Kansas City. A scientific program will be presented during the day and a dinner will conclude the event.

Regional meeting, International College of Surgeons, Madison, Wisconsin, April 26-28. All surgeons invited. Address inquiries to Dr. Arnold S. Jackson, Jackson Clinic, Madison, Wisconsin.

Annual spring congress in ophthalmology, otolaryngology, and allied specialties, Gill Memorial Eye, Ear and Throat Hospital, 711 South Jefferson, Roanoke, Virginia, April 2-7.

Nine one-day courses will be presented by the American Academy of Neurology at St. Louis, April 23-25. To be covered are neuropathology, neurophysiology, neurochemistry, electrodiagnosis, neurological diseases of infancy and childhood, injuries to the central nervous system, extra-pyramidal disorders, infections of the nervous system, and convulsive disorders. Additional one-day course on common neurological syndromes for general practitioners. Address inquries to 3501 East 54th Street, Minneapolis 17, Minnesota.

Fourth annual interim meeting of District VII, American Academy of Obstetrics and Gynecology, Peabody Hotel, Memphis, March 9-10. Scientific papers, case reports, round table luncheons, banquet on March 9. Wives invited.

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BOOK REVIEWS

Textbook of Endocrinology. By Robert H. Williams, M.D., and contributors. Published by W. B. Saunders Company, Philadelphia. 776 pages, 175 figures. Price $13.

This is an excellently written and edited text from the standpoint of applicability to clinical medicine. Each topic is developed logically from anatomy to therapy. It has the difficulty of all texts in that it is already behind the current advances in its field. Also, in condensing the material involved, the authors have been forced to oversimplify. Endocrine therapy in particular is too often presented on an empirical basis with inadequate discussion of contraindications and side effects. Definitive endocrine therapy could have been omitted with no loss to the overall contribution of the text.

This will be a good addition to the library of the practicing doctor and advanced student.—*V.E.W.*

The Relief of Symptoms. By Walter Modell, M.D. Published by W. B. Saunders Company, Philadelphia. 450 pages. Price $8.00.

This is a pleasantly written text presented almost as a philosophy of medical practice. The author has drawn upon many years of experience and personal observations to support both his therapeutic philosophy and his procedures.

The textbook would have been more valid had he not attempted to suggest definitive therapy for specific disease entities. This book is probably worthwhile reading once, especially for the third year student, but would not be recommended for addition to the library of either the student or the practicing physician.—*V.E.W.*

Booklets for Medical Societies

The American Medical Association has announced publication of two new booklets for medical society use, "Guides for Medical Society Grievance Committees" and "Report of the Survey on County Medical Society Activities." The first reviews the findings and recommendations of the special committee on grievance committees appointed by the Board of Trustees. The second includes data on society meetings, budgets, educational and scientific programs, personnel, building facilities, the work of various committees, and the extent of public relations activities.

Copies of the booklets may be obtained from the Council on Medical Service of the A.M.A., 535 North Dearborn Street, Chicago 10, Illinois.

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Kansas will still have polio problems in 1956, reports the National Foundation for Infantile Paralysis, in spite of the fact that the number of cases reported in the state in 1955 was about one-third the average number of the previous five years. The Salk vaccine has proved to be a major weapon against paralytic poliomyelitis, but it has not yet won the war against this disease.

The foundation requests the continuing cooperation of physicians in administering vaccine and in caring for patients already paralyzed and those who will be paralyzed in spite of the vaccine. Through funds contributed to the 1955 March of Dimes, the foundation gave more than 175,000 cc. of Salk vaccine to Kansas to initiate a vaccination program.

Statistics supplied by the foundation include the following: the foundation has 106 local chapters in Kansas; more than $1,910,000 has been spent by local chapters in this state for care of polio patients; 57 foundation scholarships and fellowships have been awarded to Kansas residents; grants of $825,000 from March of Dimes funds have been made to the University of Kansas for virus research and for polio prevention; in the first ten months of 1955, $19,550 in emergency aid was sent to seven Kansas chapters by the foundation.

Award of 44 unclassified life science research contracts in the fields of biology, medicine, biophysics, and radiation instrumentation was announced last month by the U. S. Atomic Energy Commission. Included was renewal of a contract for investigation of organic substances tagged with I-131 human thyroid gland in vivo, being conducted by Dr. F. E. Hoecker of the University of Kansas.

Medical School Enrollment

Although American medical schools set all-time enrollment and graduation records in 1954-1955, they may face a student recruitment problem in the next few years, according to a report of the A.M.A.'s Council on Medical Education and Hospitals. The statement is based on the fact that applications to medical schools have been decreasing for the past five years.

There were 6,977 physicians who completed work at 75 medical schools last year, the sixth consecutive year in which a record was set. This is an increase of 1,400 over the number of graduates in 1950. Seven more schools will be graduating physicians by 1960.

Total enrollment during the 1954-1955 year was 28,583, an increase of 356 over the preceding year. The entering class of 7,576 was the largest ever enrolled.

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(1) Payne, R. W.; Shetlar, M. R.; Farr, C. H.; Hellbaum, A. A., and Ishmael, W. K.: J. Lab. & Clin. Med. *45*:331, 1955. (2) Bunim, J. J.; Williams, R. R., and Black, R. L.: J. Chron. Dis. *1*:168, 1955. (3) Holbrook, W. P.: M. Clin. North America *39*:405, 1955.

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Medical Education Week

The period from April 23 to April 29 will be observed this year as Medical Education Week, a joint project of the American Medical Association, the Association of American Medical Colleges, the National Fund for Medical Education, and the American Medical Education Foundation.

This observance is planned as a means of public education, to focus attention on medical schools, to create good will and understanding, and to ease fund raising. The A.M.A. has emphasized, however, that its role in Medical Education Week will be one of public relations, not solicitation.

Increase in Hospital Room Rates

Room rates in general hospitals in the United States have increased approximately 5 per cent in the past year, according to the annual report issued recently by the American Hospital Association. The figure was based on questionnaires returned from 2,639 hospitals.

Rate figures cover the hospital room, meals for both general and special diets, general nursing service, medical records, routine housekeeping, and miscellaneous services.

The highest single room rate in the country was $35 in New York; the lowest, reported from Louisiana, was $5.00. The range for multi-bed rooms was from $23.50 in Ohio to $2.00 in Tennessee. The highest metropolitan average rate for single rooms was reported in Boston at $23.14; the lowest, $12.75 was reported for Atlanta.

Advance deposits were required from patients responsible for their own hospital bills by 47 per cent of the hospitals reporting. This practice tended to be more common among larger hospitals.

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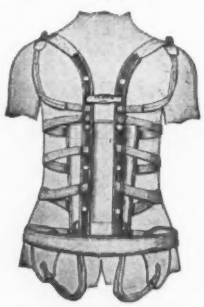

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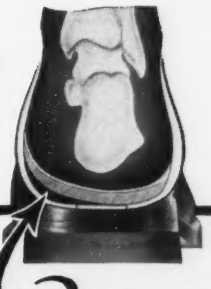

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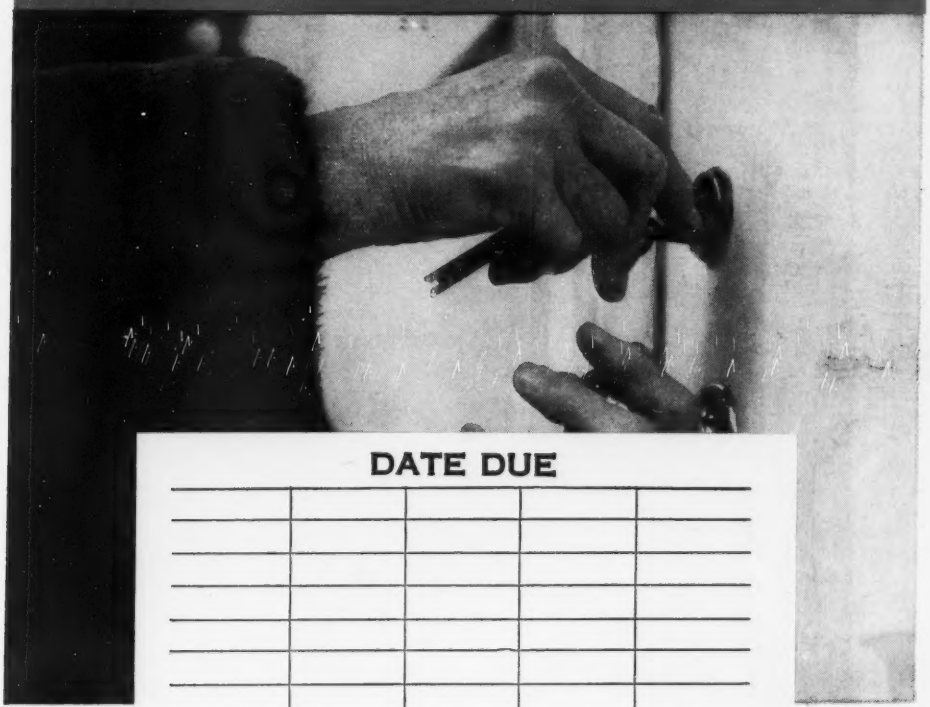

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